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Volume 35 *Number 9*

Phytopathology

AN INTERNATIONAL JOURNAL

September 1945

Official Organ of

The American Phytopathological Society

$6.25 per year, Canada $6.00 per year, United States and dependencies $6.50 per year, elsewhere

Office of Publication

Corner North Queen Street and McGovern Avenue, Lancaster, Pennsylvania

Entered at the postoffice in Lancaster, Pennsylvania, as second-class matter.

TABLE OF CONTENTS

Herbert Hice Whetzel, 1877–1944. M. F. Barrus and E. C. Stakman 659

Strumella canker of oak ..... Bailey Sleeth and Rolland C. Lorenz 671

Factors affecting the saprogenic activities of the Dutch elm disease pathogen ..... L. J. Tyler and K. G. Parker 675

Anthracnose resistance in flax ..... Charles Ray, Jr. 688

A graft-transmissible virus of sweet potato.
S. P. Doolittle and L. L. Harter 695

Movement of mosaic virus through sugar-cane seed pieces.
I. L. Forbes and P. J. Mills 705

Rhizoctonia bud rot of strawberry plants ..... J. B. Demaree 710

Fungicides in relation to scab and fruit russet of pear in the Hood River Valley, Oregon ..... J. R. Kienholz and Leroy Childs 714

The relation of the occurrence of foliage symptoms of chlorotic streak of sugar cane to the distribution of the virus in the plant.
E. V. Abbott 723

Phytopathological Notes 737

Virus diseases of guayule ..... Henry H. P. Severin
The sooty stripe disease of sorghum ..... Douglas C. Bain
Rhizoctonia foliage disease of Hevea brasiliensis. John E. Kotila
Cotton-leaf-spot Rhizoctonia and its perfect stage on sugar beets.
John E. Kotila
The longevity of the pathogen causing the wilt of the common bean ..... Walter H. Burkholder
Phoma terrestris on sugar-cane roots in Louisiana.
Fernando Carvajal

HERBERT HICE WHETZEL
1877–1944

M. F. Barrus and E. C. Stakman

The value of many a man's life may be judged most fairly by his influence on others. This intangible attribute is known fully only by those who have felt it. Professor Whetzel had a profound influence on many persons as is evident from the affection they have had for him, the testimonies they have offered, and their activity in carrying forward his suggestions. He was a unique character with extraordinary talents, some of them at least closely akin to genius. He was a great teacher, an enthusiastic naturalist, a keen investigator, an efficient organizer, a scientific leader, and a dynamic man. This is the composite judgment of his students, his colleagues, and many of his professional acquaintances. It is the judgment not merely of his friends but also of those who have considered his contributions with detached objectivity, apart from the immediate influence of his powerful personality. It is simple justice to record the debt that American phytopathology and phytopathologists owe to this indefatigable champion of his chosen scientific guild.[1]

Herbert Hice Whetzel died at his home on November 30, 1944, after being confined to his bed for three weeks by a disease against which he had valiantly fought during a period of ten years. Yet, despite the inconvenience and pain it caused him, he continued his work until nearly the end of his life.

Whetzel was born on a farm near Avilla, Indiana, on September 5, 1877, and obtained his early education at a rural school and at Avilla High School. After a period of teaching district schools, he entered Wabash College from which he graduated in 1902. While there, he came under the influence of Professor Mason B. Thomas, a Cornell man, and a most excellent teacher of botany. Probably it was due to this influence that Whetzel majored in botany. It was Thomas who obtained for him an assistantship at Cornell University and an opportunity to pursue graduate work under another great botanist, Professor George F. Atkinson. He later became an instructor in botany at Cornell and most of his time was devoted to the study of plant diseases. Before he could complete his work for a doctorate, Dean Liberty Hyde Bailey, recognizing his talents, appointed him, in 1906, an assistant professor and head of a new department of botany in the New York State College of Agriculture to be devoted to teaching and research work in plant diseases. The following year, at the earnest request of Whetzel, the name was changed to department of plant pathology. He was appointed professor in 1909 and retained this title throughout his life, although he resigned the headship of the department after an interval of sixteen years in order to devote his full time to teaching and research. He was convinced also that, with rare exceptions, no man should be allowed to head a department for

[1] Professor H. M. Fitzpatrick has prepared a personal account of Professor Whetzel's life to be published in "Mycologia" during 1945.

HERBERT HICE WHETZEL
1877–1944

more than fifteen years because long tenure deprives ambitious and able young men of the staff of an opportunity to do administrative work and he thought it might be injurious to the department or the institution itself.[2]

Professor Whetzel was first of all a teacher. His profound knowledge of phytopathology, his enthusiasm, and his interest in those willing to work attracted students to him. He had an unusual ability to present his subject clearly and logically. He taught the elementary courses himself because he believed the undergraduate students needed the clear presentation of the subject and the inspiration he was able to give them. This also gave him the opportunity to discover exceptionally gifted students and to persuade them to take graduate work. In his early years of teaching, he spent much time in preparing reading and laboratory texts of each disease used for class study, according to an outline he had originated. These were revised from time to time with the help of his colleagues in the department and two editions of the laboratory text were later published.

Whetzel took the attitude that students who registered for his courses did so because they wanted to obtain a knowledge of the subject. No record was kept of their attendance at lectures and laboratory periods; they could come or not as they wished. In order to obtain credit in a course, however, they must complete satisfactorily the work assigned. Being dissatisfied with the usual method of teaching, he devised a system whereby students select for study, within groups, certain diseases in which they are especially interested. They are provided with ample material and with the laboratory and reading texts. They may work on any disease as long as they wish, for the laboratories are always open. After completing the study of a disease, they are given an individual conference with an instructor at which the student demonstrates his knowledge of the subject by the way he uses it in solving hypothetical problems.

The graduate students, however, were not neglected. He was always available to them and his fertile mind was full of ideas relating to their problems. He was quick to give praise for their accomplishments and willing to give them full credit for their discoveries even when his suggestions made them possible. That they might know the background of their science, he published an outline of the history of phytopathology. He also gave a course in scientific German reading, using a method he devised which enabled the student to acquire a proficiency in reading the language within a year. He did whatever was possible to fit them for their profession and they left for their own fields of activity with a deep sense of gratitude and an enduring attachment to him.

But Whetzel's influence extended far beyond his own classroom. During the period of his greatest vigor, he made a two-fisted fight for better teaching and he expounded his pedagogical ideas far and wide with a zealousness born of intense conviction. To him the individual student was the most important element in education. He often said "Nobody ever

[2] Jones, L. R. Whetzel resigns headship—deplores present system of administrative tenure. Phytopath. **12**: 499–500. 1922.

taught anybody anything." His idea was that a teacher was good not so much because of what he transmitted to students but because of what he got them to learn. This educational doctrine was not new but Whetzel really made it work. Students who had passed his individual-effort courses stuck out their chests a little farther and elevated their chins a little higher: they had acquired confidence in their own intellectual power, in their ability to find out about things by themselves. Insistence on individual work, on respect for facts, on refinement of concepts, on precise terms and definitions, and on organization of knowledge was the keystone of Whetzel's pedagogical creed. And, born agitator that he was, Whetzel at least jarred many other teachers out of complacency and, in his peppery way, injected enough intellectual irritants into their minds to make them think about improving their own methods even if they did not accept his.

Whetzel always wanted farmers to have the best and latest information on disease control. In the early years of the department's life, he spent considerable time in preparing exhibits and in attending fairs. He addressed farmers many times at state and county meetings and used every opportunity to impress upon them the importance of controlling diseases. Once, he gave a talk on apple scab, using charts, at a night meeting of the state horticultural society. After the meeting, the extension director who is also a fruit grower, remarked, "If anyone had told me I would sit on a hard bench in a hot crowded room for two hours listening to a man spout about apple scab, I would have told him he was crazy. But I did and I liked it and I learned a lot." During those years, Whetzel was well known and well liked by New York farmers. They had confidence in him even to the extent of being willing to contribute money for research work on their disease problems when he proposed it.

He believed in the socialization of his science. But he was hard-headed enough to maintain that plant growers would appreciate and use most effectively those services for which they paid in hard cash. He was confident that eventually even the private plant doctor would find a field of usefulness. At a time when many agricultural scientists still looked askance at funds offered by commercial organizations for research, Whetzel boldly encouraged and advocated the industrial fellowship plan and made it work. Not only corporations but also growers' organizations were brought into partnership with the University in the investigation of plant-disease problems. These fellowships made it possible for qualified students to undertake graduate work who would have been unable to do so without financial assistance and they have resulted in the accomplishment of much practical experimental work that, without them, would not have been done.

The generation of plant pathologists who were in process of professional development while Whetzel was in his prime probably were most impressed by his Kühnian attitude: the utilization of basic information in plant-disease control. Certainly Whetzel and his associates at Cornell were in the vanguard of those pioneers who began to emancipate practical plant pathology

from a murky empiricism and place it on a sound scientific basis. He appreciated the possibilities of improvement in control methods and materials. As soon as he learned of Cordley's results with lime-sulphur as a fungicide, he had a fellowship organized to study further the possibilities of this material in controlling diseases in New York State. He championed dusting as a substitute for spraying in the early days of its use for disease control and defended the method in the face of severe criticism when its limitations became apparent, for he believed that, with improvement in quality of dusts and in machinery for applying it, this method of protection would be generally used.

Despite his advocacy of exacting standards of experimentation in a very practical field of endeavor, Whetzel remained at heart a naturalist, a collector, and a mycologist. His intimate acquaintanceship with growing things in the woods and fields was not only the envy but also the despair of many who had the desire but not the ability to master successfully so wide a range of pure and applied mycology.

During his later years, he devoted much time to the study of the Sclerotiniaceae in which he was interested throughout most of his professional life. He collected assiduously every spring and also received specimens from other mycologists knowing his interest in this group. Many papers relating to the genera and species have been published by him and by his students. At one time he planned to prepare a monograph of the North American species but, as his work progressed, he recognized the need for a more detailed study of the new species discovered and for erecting new genera before it would be possible to monograph them. Realizing finally that his time was short, he started to prepare a characterization of the new family Sclerotiniaceae and a synopsis of the fifteen genera he had included in it. Although it is unfortunate that he was unable to complete this before his death, he did succeed in greatly increasing our knowledge of this important family of fungi.

Whetzel's researches cover a wide range in phytopathology, as the list of his publications indicates. While he was still an instructor in botany, he published three important bulletins on plant-disease control. During his lifetime he was active in studying diseases of many kinds of plants, including medicinal, ornamental, and wild plants, and in the study of fungicides and control measures. And yet to many pathologists Whetzel's critical faculty may have been even more stimulating than his own researches. It is a great misfortune that he published so little of his vast store of information, but his "Are you sure of that?"; his "I'll bet you a cigar that I can prove you wrong"; and his less contentious "Did you ever try this—?" in meetings, in his favorite "rag-chewing sessions," and in correspondence suggested many a new line of attack on obscure problems and led to many a discovery. Dynamic he was, not only in teaching but also in research, and he added to his zeal for both a profound faith in the service of organized plant pathology in plant production and plant protection.

The plant pathology herbarium at Cornell University is a monument to Whetzel's interest in fungi. His own collections formed its nucleus to which he and others have added many specimens since. It now contains more than 31,000 specimens besides private and special collections and purchased exsiccatae. Since 1922, when he first collected in Bermuda, he had been active in building up collections from the Tropics. He made three trips to Puerto Rico and one to Venezuela during which he collected extensively with other mycologists. He collected fungi wherever he went and stimulated his students to do likewise, with the result that specimens have come from many countries and especially from Latin-America.

Whetzel was one of the charter members who organized the American Phytopathological Society in 1909 and was one of its guiding spirits until his physical condition prevented him from attending meetings. And he worked hard to make the Society useful not only to its members but to the advancement of science as well. He was one of the promoters in the organization of the Mycological Society of America and took a leading part in its affairs as well as contributed to its official journal.

Gardening was another activity that gave him much pleasure. He grew many kinds of plants, including new varieties of vegetables and fruits. His greatest interest was wild and cultivated varieties of flowering plants, especially unusual and rare species and varieties. He delighted to show these to the many persons attracted to his garden and generously gave plants and seeds to those who indicated an interest in them. From this garden also came many prized specimens of plant diseases he had discovered and maintained.

A many-sided man, Whetzel was an intensely interesting character. It is difficult to suppress an impulse to write about his human qualities because, to those who knew him best, his sterling character and his capacity for enduring friendship command respect and admiration quite as much as his intellectual contributions. His friends will always keep something of "Whet" within themselves. But this is being written not as a eulogy but as an evaluation of Whetzel's services to Plant Pathology. To say that he was "Prof" to many aspiring young pathologists who never entered his classroom would alone be high tribute. But, it is even higher tribute to say that he was still "Prof" to many of his professional associates who fought him hard on certain issues. For "Prof" was a rugged, aggressive, and game fighter. He fought for his convictions and came into an argument with crackling energy and a determination to rush his opponents through the ropes. He never held resentment long against anyone and never spoke maliciously of others.

No longer will his footsteps be heard in the corridors of the University nor his cheery "Howdy" greet his friends as in the past. His voice will not be raised in arguments at gatherings he loved to attend nor will his friendly counsel guide his students anymore. Yet, in the hearts of all who knew him will remain the memory of the man, and his influence will prevail for many years to come. His life was full and rich because he lived it with others.

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STRUMELLA CANKER OF OAK

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(Accepted for publication March 26, 1945)

The distribution, host species, and symptoms of the canker disease attributed to the fungus *Strumella coryneoidea* Sacc. have been described by Heald and Studhalter[2] and Bidwell and Bramble.[3] These investigators, on the basis of association, determined the causal fungus to be *Strumella coryneoidea* Sacc. Heald and Studhalter state that they secured successful inoculations in the field. However, they do not indicate the source of the cultures or the methods used, or give data on number of successful inoculations or illustrations of induced infections. Because of the lack of definite information concerning inoculations and the production of infection and cankers, it was considered advisable in the course of general work on control of the Strumella canker disease to inoculate young oak trees with the fungus, which had been consistently isolated from Strumella cankers, in an attempt to establish infection and produce typical cankers.

INOCULATIONS

The inoculations herein reported were made in the fall of 1934 in the Logan State Forest in the vicinity of State College, Pa., and the following year in Connecticut. Saplings of the following species were inoculated: White oak (*Quercus alba* L.), chestnut oak (*Q. montana* Willd.), red oak (*Q. borealis* var. *maxima* (Marsh.) Ashe), scarlet oak (*Q. coccinea* Muench.), and black oak (*Q. velutina* Lam.). The trees inoculated ranged from dominant to suppressed types. Most of the inoculations were made with cultures that had been isolated from typical Strumella cankers from different oaks (Table 1), but a few were made with masses of spores from freshly collected fruiting bodies of *Strumella coryneoidea* found on dead oak branches.

Live branches $\frac{1}{4}$ to 3 inches in size were inoculated. Some branches were girdled a month before they were inoculated and some were girdled when inoculated. Inoculations were made either above or below the girdle and in some cases both above and below. The purpose of the girdle was to weaken, but not kill, the branch. It was thought that a branch thus weakened by girdling might provide conditions favorable for the establishment of infection and subsequent canker formation. Also, inoculations were made on ungirdled branches and trunks of very small trees. The inoculations per tree ranged from 3 to 6. The inoculum, other than spores from fruiting bodies, consisted of autoclaved rice on which the fungus had grown.

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[2] Heald, F. D., and R. A. Studhalter. The Strumella disease of oak and chestnut trees. Pa. Dept. Forestry Bull. 10. 1914.

[3] Bidwell, C. B., and W. C. Bramble. The Strumella disease in southern Connecticut. Jour. Forestry 32: 15–23. 1934.

The technique of inoculation was similar to that described by Hahn and Ayres.[4] The area around the point to be inoculated was carefully washed with 95 per cent alcohol. A small short-handled chisel, sterilized by dipping in alcohol and flaming, was used to make an incision, approximately 1 inch in length, parallel to the long axis of the trunk or branch and just through the bark. The bark was raised slightly on one side of the slit by pushing the tip of the chisel under the bark and giving a slight pry on the handle of the chisel. In the opening thus made a small quantity of rice inoculum was placed. In a few cases where masses of spores were used the inoculation procedure was essentially the same. A small pad of sterilized cotton was placed over the inoculated wound and bound firmly in place by a cotton

TABLE 1.—*Reisolations from Strumella inoculations made at Sand Spring, Logan State Forest, Pennsylvania*[a]

Source of cultures	Oaks inoculated and number of inoculations yielding Strumella when cultured							
	White oak		Chestnut oak		Black oak		Red oak	
	+[b]	−[b]	+	−	+	−	+	−
White oak	2	0	2	0	2	0	1	0
White oak[c]	2	2	1	1	1	0	1	0
Chestnut oak	3	0	2	0	1	0	1	0
Scarlet oak	5	1	1	1	1	0	1	0
Black oak	2	1	1	1	1	0	1	0
Total	14	4	7	3	6	0	5	0

[a] The Strumella fungus was recovered in all cases in invaded tissue beyond the point of inoculation. All inoculations made with sterile rice inoculum gave negative results.

[b] + indicates infection; − indicates no infection.

[c] This culture was from a pustule on a diffuse Strumella canker. The other cultures used were from cankered tissue of typical "cat-faced" cankers.

string. In a few cases in which no cotton was used, the wound was wrapped with the cotton string. The tight binding forcibly brought the inoculum in contact with the ruptured bark tissues. Finally, the wound was carefully wrapped with a 3- to 5-inch band of waxed paper or parchment paper, which was fastened at the top and bottom with commercial zinc oxide adhesive tape, or mechanic's tarred tape. The top of the paper band was bound tightly to the branch or stem with tape. In most cases this made an effective waterproof seal. The wrapping protected the incisions from contaminations and prevented excessive drying of the ruptured and inoculated tissues. Checks were made in a similar manner, in which sterile rice was used for inoculum.

RESULTS

A small number of inoculated branches were removed at various intervals to determine whether the Strumella fungus had become established in the inoculated tissues and to note the progress of infection, if any. In May, 1935, 6 months after inoculation, *Strumella* was recovered from 12 in-

[4] Hahn, Glenn Gardner, and Theodore T. Ayers. Failure of Dasyscypha willkommii and related large-spore species to parasitize Dougles fir. Phytopath. 28: 50–57. 1938.

oculated branches out of 14 removed for examination and culturing. The checks were negative. The fungus had established itself in the wounded tissue and had advanced a slight distance into the uninjured live tissue adjacent to the incision (Fig. 1, A). These results indicate that the fungus was able to spread to some extent during the winter.

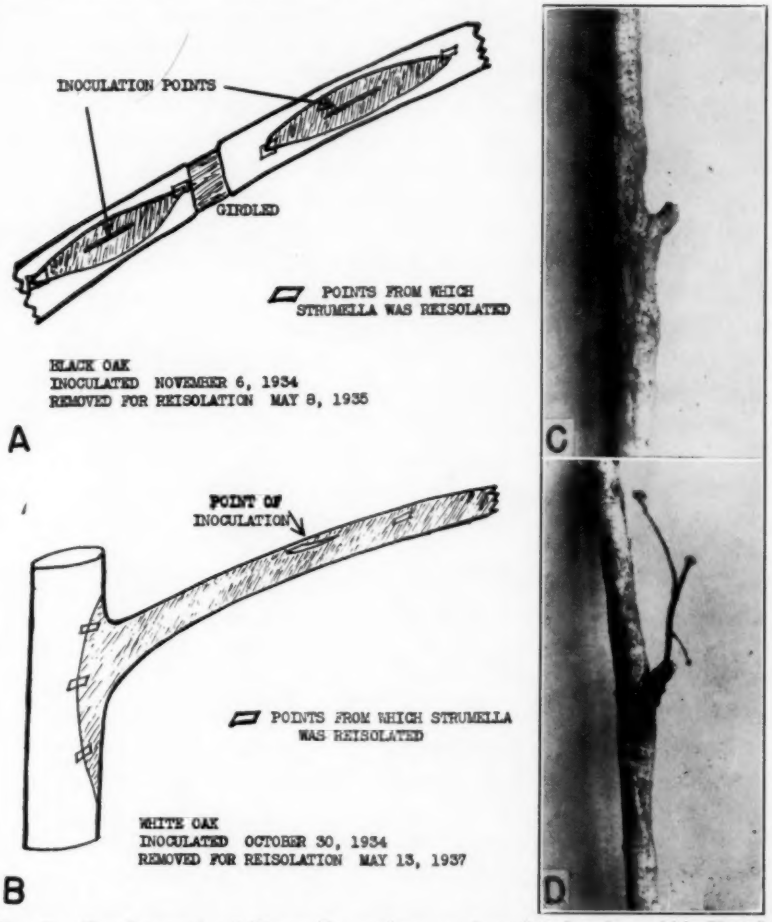

FIG. 1. Development of Strumella cankers on inoculated oaks. (A) Sketch of a girdled black oak branch 6 months after it had been inoculated (shaded portion indicates dead area). (B) Sketch of a white oak branch 2½ years after it had been inoculated. The fungus has progressed down the inoculated branch into the trunk. Strumella canker development 3 years after inoculation, (C) on chestnut oak and (D) on white oak.

A little over a year after the inoculations were made, December, 1935, a few more inoculated branches were removed. *Strumella* was recovered in 9 cases out of 11 inoculations made from Strumella cultures. The fungus was recovered at considerably greater distances from the point of inoculation than from those examined in May. Whether the fungus progressed at a uniform rate in the uninjured tissues during the 7-month interval, or made rapid progress after cessation of tree growth in late summer is not known. In a number of cases the fungus had killed the inoculated branch by

girdling. As might be expected, the longitudinal advance of the fungus was much greater than its circumferential progress.

To check on the inoculations made with Strumella spores from sporodochia on small dead oak branches 8 inoculated branches were removed in December, 1935. In only one instance was *Strumella* isolated from the tissue adjacent to the inoculated incision. This may have been a chance infection from some other source. However, if a sufficient number of successful inoculations could be secured by this method it would establish definitely that the spores thus produced in nature do germinate and are a potential source of inoculum for natural infections. Successful germination of Strumella spores has not been reported to the writers' knowledge.

By May, 1937, many of the inoculated branches were showing definite indications of canker formation. In some cases, two definite somewhat concentric callus rings had developed around the point of inoculation. At this time 8 inoculated branches were removed, 5 of which yielded *Strumella*. The fungus had killed the inoculated branch in most instances and had progressed down the branch and invaded the trunk in some cases (Fig. 1, B). Two small trunk cankers resulting from branch inoculations are shown in figure 1, C and D. It seems most likely that natural trunk cankers develop in a similar manner, in which infection enters the trunk from a small branch killed by the invading fungus. This would account for the occurrence of a dead branch stub in the center of the canker, which tends to develop in an ellipsoidal fashion.

Late in October, 1937, six inoculated branches with definite canker formation were removed. All 6 cankers yielded *Strumella* in culture.

Results similar to those secured in Pennsylvania were obtained from inoculations that were made in a plot in Connecticut in October, 1935. Three scarlet oak and three red oak branches that had been inoculated with *Strumella* and that had typical lesions were removed from the Connecticut plot in June, 1937. All 6 inoculated branches yielded the fungus upon culturing.

Successful cross inoculations were made on both girdled and ungirdled branches among a few oak species (Table 1) in the Pennsylvania plot. There was no indication that an isolate from one oak species was more virulent than any other. Unfortunately, observations could not be continued for a sufficient length of time to secure conclusive data on this point. However, the results do indicate that the fungus is not a highly specialized parasite.

SUMMARY

Successful inoculations and initial canker development were secured on various oaks with a fungus, identified as *Strumella coryneoidea*, which had been isolated from typical Strumella cankers.

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FACTORS AFFECTING THE SAPROGENIC ACTIVITIES OF THE DUTCH ELM DISEASE PATHOGEN

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(Accepted for publication April 2, 1945)

The Dutch elm disease fungus (*Ceratostomella ulmi* (Schwarz) Buisman) lives as a saprogen for a considerable part of its normal life cycle. During this phase the organism is subject to the influence of a number of environmental factors that may greatly affect its existence. Information about the effects of different factors is rather scarce in the literature and so the purpose of this paper is to help elucidate the effects of temperature and moisture on the saprogenic growth and survivability of *C. ulmi*.

LITERATURE REVIEW

Ledeboer (4) reported that *Ceratostomella ulmi* grew fairly well on artificial media at 8.5° C.; the optimum temperature was 25° C. and the maximum about 34° C. May (5) found that, for growth on the media used, the minimum temperature was between 5° and 9° C., the optimum between 22° and 27° C., and the maximum about 36° C. He stated, further, that temperatures from 10° to 35° C. had no differential effect on the relative abundance of coremial fruiting structures produced by the fungus.

True and Slowata (9) found that the Dutch elm disease pathogen survived in naturally diseased cut elm wood at least 20 months under some field conditions. In their test, diseased wood fully or partly shaded from the sunlight all or part of the time maintained the organism in a living state longer than did that exposed to more or less constant sunlight and desiccation.

Buisman (2) reported that spores in dried masses withstood a 2-month drying period in Petri dishes at room temperature. Moses and Hoffman (6) exposed beetles (*Scolytus multistriatus*), surface-infested with spore masses, to a series of temperatures that ranged from −10° F. (−23.5° C.) to 70° F. (about 21° C.). The fungus survived about 3 months at 70° F. on 60 per cent of the beetles while after 2½ years it was recovered from all the beetles that were exposed to −10° F. May (5) reported 51° C. as the thermal death point for spores in water and 57° C. for those exposed on dry glass rods.

Tyler *et al.* (10) reported that high air humidity favored infection of small potted elms inoculated by introducing spores into wounds that were made by noninfested adults of *Scolytus multistriatus* Marsh.

METHODS AND MATERIALS

Six cultural races of *Ceratostomella ulmi*, all pathogenic to the American elm, were used as noted in the following studies. They originated as sector variants in mass isolates from diseased elms and the stock cultures were

started from single spores. Distinct differences in cultural characters such as those noted by Walter (11) existed among the races. Some races produced coremia abundantly when grown on nutrient agar and on elm wood, others produced none, while still others were intermediate in this character.

The nutrient solution commonly used in spore germination tests consisted of a 0.9 per cent solution of maltose (technical grade) in distilled water; it was sterilized by means of a suitable Berkefeld filter and usually tested about pH 6.2.

The artificial, nutrient medium used was potato-dextrose agar. It consisted of the extract obtained by boiling 390 grams of peeled Irish potatoes for 20 minutes in a quantity of distilled water, 20 grams of dextrose, and 18 grams of shredded agar made up to one liter with distilled water. A quantity of this medium sufficient for a given experiment was prepared in one batch and autoclaved for 20 minutes at 15 pounds pressure. The pH concentration of the nutrient agar after autoclaving usually was about 5.8.

Diseased elm wood used in some of the temperature and humidity experiments was obtained from 3- to 4-year-old potted elms that were artificially inoculated with chosen races of the pathogen. Before the disease had killed the trees, branches $\frac{1}{2}$ to 1 inch in diameter were cut from them and wrapped in waxed paper then stored at 5° C. until needed.

Temperature Studies

Spores from coremia[1] approximately 10 to 15 days old were suspended in the maltose solution. Drops of the spore suspension were placed on sterile glass micro-slides in inverted glass moist chambers. The density of the spores in suspension was adjusted so that about 50 to 75 spores were present in the microscopic field as seen with the dry, high-power objective of a compound microscope. Usually the spores were exposed to the desired temperature 10 to 13 hours before counts were made.

Petri dishes containing the solidified nutrient agar were planted by transferring to each a 4-mm.-wide disc cut from a 4-day-old agar culture of *Ceratostomella ulmi*. Planted plates used in the temperature studies were exposed for 8 days and during this time they were kept within sealed glass chambers to prevent excessive moisture changes in the medium.

The effect of temperature on development of coremia on wood from diseased trees was studied by two culture methods. For one, discs of diseased elm wood about $\frac{1}{2}$ inch in diameter by $\frac{1}{8}$ inch thick were cut from stored material and placed on 1.5 per cent non-nutrient agar in Petri dishes. Triplicate plates containing the discs were placed in Stender chambers whose lids were sealed with vaseline. Such chambers prevented excessive desiccation of the cultures held at the higher temperatures. For the other method, discs of wood similar to the above were placed on sterilized, flat cotton lamp wicks that were drawn through $\frac{5}{8}$-inch-bore glass tubing. The tubing was first cut in lengths such as would fit, when supported horizon-

[1] These spores are termed coremiospores in the paper.

tally, into a Stender chamber 5¼ inches in diameter by 2½ inches deep. One end of the wick dipped into sterilized water in a wide-mouth bottle inclosed within the chamber, the other end extended almost to the opposite end of the tube which was loosely plugged with nonabsorbent cotton. The moist wicks kept the moisture content of the wood at the proper level. Before the fungus-invaded discs of wood were placed in the culture chambers the latter were sterilized by dry heat. After the wood discs were placed within, the chambers were sealed with vaseline and placed at the desired temperatures. The cultures were examined periodically by means of a binocular microscope.

Humidity Studies

To study the effect of air humidity on germination, coremiospores were suspended in maltose solution. The spore suspension was sprayed on surface-sterilized glass micro-slides with an atomizer that was sterilized in boiling water. The sprayed slides were allowed to dry for about 3 hours; afterward they were placed on racks in Stender chambers over a series of salt solutions each of which exerted a different vapor pressure. Each chamber contained 4 slides, 2 with the dried spore suspension facing downward and 2 facing upward. All culture chambers in the series were sealed and exposed at 24° C. After exposures of 24 and 48 hours the slides were examined with a compound microscope.

The effect of air humidity on the germination of spores and on the growth of hyphae on elm wood was studied. Discs of elm wood were cut from fungus-free wood, and from diseased wood. The fungus-free wood discs were sprayed with coremiospores suspended in sterile distilled water. Discs of both kinds were air-dried for 3 hours and some of each were placed in each of four desiccators. Humidity of the air in each desiccator was controlled by first passing it through a suitable salt solution and then through the desiccator. Salt solutions in the towers through which the air was passed were changed every 72 hours. The treatment was continued for 14 days after which the discs were carefully examined. The absence of mycelia was considered as proof of the inability of the organism to grow under the conditions imposed.

Survival Studies

The effects of higher temperatures and of moisture on the survivability of *Ceratostomella ulmi* were studied using discs of diseased elm wood like those described. By means of a simple irrigation device, wood discs inclosed in sealed Stender chambers were kept moist but not wet. In similar chambers, without the irrigation device, other wood discs were allowed to dry. At chosen intervals 5 moist and 5 dry discs were selected at random from chambers exposed at 30°, 33°, 36°, and 39° C. The discs were planted on nutrient agar immediately following their removal from treatment chambers and growth of the fungus was later recorded.

For a further test of the survivability of the fungus, branches 3 to 5 inches in diameter were obtained from a naturally inoculated diseased elm

and these were cut into a number of 3-inch-long sections without removing the bark. The exposed wood surfaces of about one-half the number of sections were covered with paraffin to prevent excessive changes in water content; those of the remaining sections were left unprotected. Seven lots were prepared and each was stored as shown in table 7. At intervals of two months a sample was taken from each lot. Tissue with fungus discoloration was dissected from each sample; some of it was planted on acidified and some on nonacidified potato-dextrose agar and growth was later recorded.

RESULTS

The data presented are representative results obtained by repeating each of the different experiments several times except for those obtained from the 2-year survivability test which was not repeated.

Effect of Temperature

Spore germination. The coremiospores of *Ceratostomella ulmi* germinated best at 27° C.; at this temperature the percentage of spores germinated

TABLE 1.—*Effect of temperature on the germination of coremiospores of Ceratostomella ulmi*

Race	Per cent germinated[a]										
	Temperature, °C.										
	3	6	9	12	15	18	21	24	27	30	33
B	0	0	0	0	Tr[b]	Tr	75.6	93.4	97.0	94.0	Tr
C	0	0	0	Tr	Tr	Tr	73.6	96.1	98.6	95.6	Tr

[a] After an incubation of 40 hours the cultures held at 18°, 15°, 12°, 9°, 6° and 3° C. were budding; budding was profuse at 18° and tapered off to a bare trace at 3° C.

[b] Tr = trace.

for each of two representative cultural races was respectively, 97.0 and 98.6. In general, spores germinated well from 21° to 30° C., inclusive, during 13 hours. Very few spores germinated at 12°, 15°, 18°, and 33° C. At 18° C. and lower temperatures the spores tended to bud in a yeast-like manner; budding was profuse at 18° C. and scanty at 3° C. (Table 1).

The organism was rather capricious in regard to spore germination and at least three factors appeared partly responsible. First, probably because of inherent differences the spores of some races germinated in greater numbers than did those of others. Second, spores that were taken from 10- to 15-day-old coremia usually germinated better than those from younger or older fruiting structures. Third, the nutrient medium was important because spores germinated much better in a solution of technical grade maltose than they did in a solution of chemically pure maltose. Since pyridoxine (3, 7, 8) and certain minor elements[2] appear to be essential to the growth

[2] Pope, Seth A. Unpublished data. Plant Pathology. Cornell University. Ithaca, New York.

of *Ceratostomella ulmi* it is possible that these or other similar growth-promoting substances existed in the technical grade maltose but were absent from the chemically pure sugar.

Growth of mycelium. Vegetative growth of the fungus on potato-dextrose agar was more rapid at 27° C. than at any of the other temperatures (Figs. 1 and 2). The temperature range favoring generally rapid growth on solid media extended from 21° to 30° C., inclusive, and it is recalled that these limits marked the range most favorable for spore germination. Growth occurred at about 3° and 33° C., but not at 1° C. nor at 36° C. Decrease

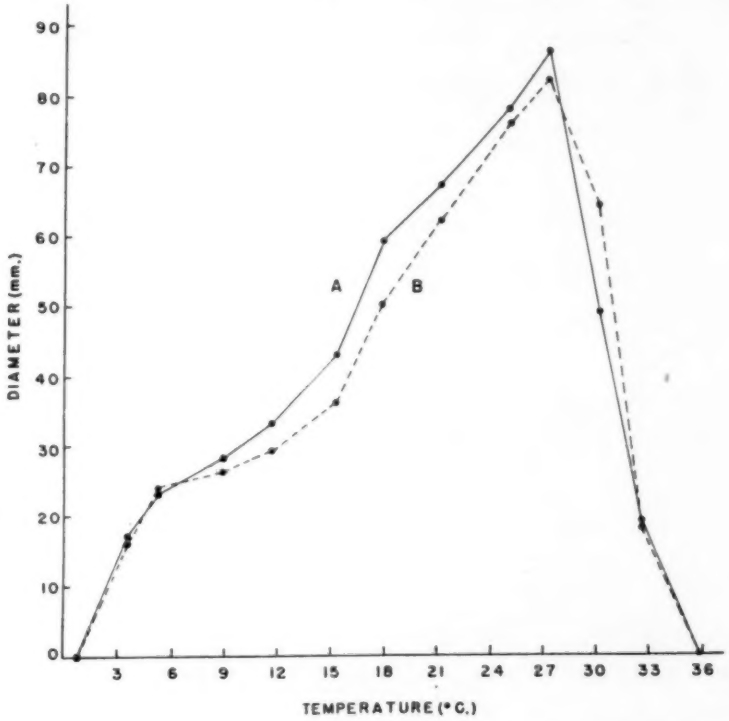

FIG. 1. The effect of temperature on the growth of *Ceratostomella ulmi*, cultural races A and B, on potato-dextrose agar.

of the growth rate at temperatures above 30° C. was extremely abrupt while the depression was gradual from the optimum of 27° C. down to about 3° C. (Fig. 1).

Colonies of the fungus grown at 12° C. or lower, and at 30° C. were distinctly white in contrast to the pale greyish cream color of those grown at more favorable temperatures. Conidiophore development of the "Cephalosporium type" was pronounced at favorable temperatures but colonies grown at temperatures less favorable were mostly without aerial conidiophores. The latter type of colony was composed of compact, irregular hyphae and many yeast-like cells. The fungus races used responded simi-

larly to temperature and each maintained its distinctive cultural characters at temperatures in the range that favored growth.

Growth of coremia. The optimum temperature for development of coremia on elm-wood discs mounted on non-nutrient agar was about 24° C. (Table 2). In cultures examined after they had grown for 4 days coremia had developed in those of race C, from 21° to 30° C., but not in those of other races. In thirty days coremia formed in cultures of race C at all temperatures from 3° to 30° C., but not at 33° C.; there were very few formed during this period at 6° C. and lower. Races F and D failed to produce coremia at any temperature tried and this result conforms with all past observed behavior of these races in relation to the production of coremia.

TABLE 2.—*Effect of temperature on coremial development by three different races of Ceratostomella ulmi. Diseased wood mounted on non-nutrient agar*

Temp., °C.	Relative abundance[a] of coremia after					
	4 days			30 days		
	Race			Race		
	F	C	D	F[b]	C	D[d]
3	−	−	−	−	−+−[c]	−
6	−	−	−	−	−+[c]	−
9	−	−	−	−	++[c]	−
12	−	−	−	−	+++	−
15	−	−	−	−	+++	−
18	−	−	−	−	+++	−
21	−	+	−	−	+++	−
24	−	++	−	−	+++	−
27	−	−+	−	−	+++	−
30	−	−+	−	−	+++	−
33	−	−	−	−	−	−

[a] Number of coremia per wood section: −=none; −+−=1 to 5; −+=6 to 10; +=11 to 20; ++=21 to 30; +++=more than 30.

[b] Mycelium present at all temperatures but very scanty at 3°, 6°, 9° and 33° C.

[c] Short coremial fundaments.

[d] Mycelium present at all temperatures except 33° C. and very scanty at 3°, 6°, 9° and 12° C.

Results essentially similar were obtained when discs of diseased elm wood were placed on moist cotton wicks (Table 3). In addition to race C, used in the first experiment, two other coremium-forming races, B and E, were used. Temperatures of 6°, 3°, and 1° C. appeared to inhibit the formation of coremia by the two latter races and in this respect they differed from race C which had the capacity to form coremia even at 3° C.

The temperature optimum for coremium development did not appear to be the same as that inducing the highest percentage spore germination and the most rapid mycelial growth (cf. Table 1, Fig. 1, and Table 2). Visual evidence of this for race B is in figure 2; the dark concentric rings in cultures held at 21° and at 24° C. were due to the presence of coremia. On potato-dextrose agar the coremia formed best at 24° C., fairly well at 21° C., but they formed sparingly or not at all at the other temperatures during the 8-day exposure.

TABLE 3.—*Effect of temperature on coremial development by three different races of Ceratostomella ulmi. Diseased wood mounted on cotton wicks*

Temp., °C.	Relative abundance[a] of coremia after					
	4 days			20 days		
	Race			Race		
	B	C	E	B	C	E
1	−	−	−	−	−	−
3	−	−	−	−	−	−
6	−	−	−	−	−	−
9	−	−	−	−	−	−
12	−	−	−	−	−+−	−+
15	−	−	−	−+−	−+	+
18	−	−	−	−+	+	++
21	−	−	−	++	+	++
24	+	−	−+	++	++	+++
27	−+−	−+−	−	++	++	+++
30	+	−	−	+++	++	+++
33	−	−	−	−	−	−

[a] Number of coremia per wood section: − = none; −+− = 1 to 5; −+ = 6 to 10; + = 11 to 20; ++ = 21 to 30; +++ = more than 30.

After 80 days the lowest temperature at which coremia were formed by each of the different races was: B, 12° C.; C, 3° C.; E, 9° C. None of the races formed coremia at 33° C. but there was very scanty growth of mycelium.

Effect of Humidity

Spore germination. Coremiospores of *Ceratostomella ulmi* germinated within 24 hours on glass slides at relative humidities of approximately 98 and 100 per cent (Table 4). Those exposed at approximately 95, 93, 90, and 85 per cent relative humidity failed to germinate. At the end of 48 hours spores that germinated at 98 and 100 per cent relative humidity respectively had formed long, vigorously branching hyphae.

Mycelial growth and fruiting. The growth of *Ceratostomella ulmi* in and on elm wood was markedly influenced by air moisture (Table 5). In

TABLE 4.—*Effect of humidity on germination of coremiospores of Ceratostomella ulmi*

Per cent relative humidity	Degree of germination[a]			
	Slide surface			
	Lower		Upper	
	Hours' exposure		Hours' exposure	
	24	48	24	48
100	+	++	+	++
98	+	++	+	++
95[b]	−	−	−	−
90	−	−	−	−

[a] ++ = excellent germination with long branching hyphae and numerous conidia; + = good germination with less branching and fewer conidia; − = no germination.

[b] Negative results were obtained also at 85 and 93 per cent relative humidity.

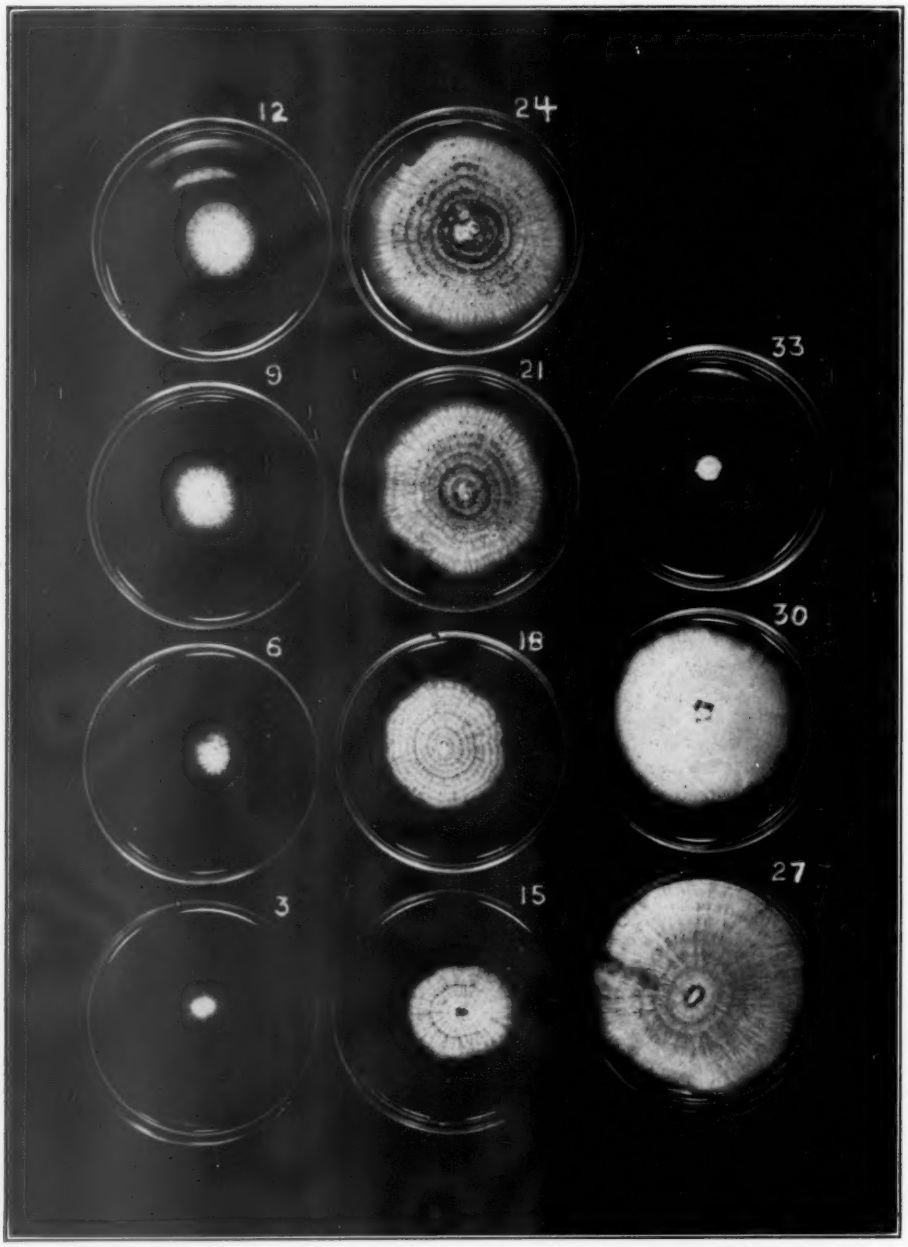

FIG. 2. The effect of temperature (°C.) on the growth of *Ceratostomella ulmi*, race B, on potato-dextrose agar.

general, growth of the fungus was good in an atmosphere whose relative humidity was about 98 per cent or higher, both on healthy elm wood planted with spores and in invaded or diseased wood. At 96 per cent relative humidity and lower the organism failed to develop on healthy elm wood planted with spores, but it did grow out from diseased wood. Coremia developed on diseased wood at 96 per cent relative humidity and higher but on elm wood planted with spores they did not develop at a relative humidity lower than 98 per cent. From these observations it appears that if the organism exists as hyphae in the wood it is far more likely to grow when moisture of the surrounding air is lowered than it is if in the form of scattered spores on the outer surfaces of wood.

TABLE 5.—*Effect of air humidity on the growth of Ceratostomella ulmi on elm wood. Exposed at room temperature.*

Per cent relative humidity	Type and relative amount of growth[a]					
	Planted elm wood			Diseased elm wood		
	Coremia	Conidia	Mycelia	Coremia	Conidia	Mycelia
92	−	−	−	−	−+	−+
96	−	−	−	−+[b]	+++	+
98	+[b]	+++	+	++[c]	+++	++
100	−+	+++	+	+++[c]	+++	++

[a] Coremia and conidia: −= none. −+= very few. += few. ++= many. +++= numerous.
Mycelia: −= none. −+= very sparse. += fairly abundant. ++= abundant.

[b] Small coremial heads.
[c] Large coremial heads.

Effect of Temperature and Moisture on Survival

Temperature and moisture exerted a pronounced influence on the survivability of *Ceratostomella ulmi*. In small discs of diseased wood that were allowed to dry the fungus survived 8 and 6 days at 30° and 33° C., respectively, while in wood kept moist it was still alive at the end of 14 days (Table 6). In moist wood at these temperatures it is likely that the organism would live much longer. At 36° and at 39° C., *C. ulmi* lived in wood kept moist for 8 and 4 days respectively, while in wood allowed to dry the survival periods were 4 and 3 days respectively. Thus moisture aided *C. ulmi* to survive in wood at higher temperatures, but this protective effect rapidly diminished with increase in temperature above 33° C.

In another experiment coremiospores were suspended in sterile, distilled water and sprayed on hot-air-sterilized elm wood. The planted wood was placed in test tubes plugged with nonabsorbent cotton and capped with cellophane to prevent excessive accumulation of moisture in and on the wood. Some of the cultures were stored constantly at −10° C.; others were held constantly in a room whose temperature fluctuated between 12° and 20° C. At chosen intervals wood bearing spores was removed from the tubes, the

TABLE 6.—*Effect of temperature and moisture on the survival of Ceratostomella ulmi in small discs of diseased wood*

Temp., °C.	Days surviving in diseased wood[a]													
	Moist							Dry						
	2	3	4	6	8	10	14	2	3	4	6	8	10	14
30	+	+	+	+	+	+	+	+	+	−+	−+	−+	−	−
33	+	+	+	+	−+	+	+	+	−+	−+	−+	.....	−	−
36	+	+	−+	−+	−+	−	−	+	−+	−+	−	.....	−	.....
39	+	−+	−+	−	−	−	.....	+	−+	−	−	−	.....	.....

[a] + = organism survived in all samples from given lot.
−+ = organism survived in part of samples from given lot.
− = no survival; ..... indicates no test.

spores were washed from the wood and suspended in maltose (1.5 per cent) solution that was acidified to prevent the growth of bacteria. Drops of the spore suspension were placed on glass slides mounted in moist chambers that were kept for 12 to 14 hours at 25° C. The viability of spores from each of the treatments rapidly declined. At the end of one month about 8 per cent of the spores from each treatment were germinable. After 4½ months about 2 per cent of the spores exposed at −10° C. were still viable, while those exposed at 12° to 20° C. failed to germinate at the end of 1½ months. About 5 per cent of the spores taken from coremia that were allowed to dry at the same temperatures retained their germinability for 4½ to 5 months.

In larger sections of wood cut from naturally diseased elm, *Ceratostomella ulmi* survived as long as two years (for the duration of the test) under conditions that prevented rapid desiccation (Table 7). On the other hand, the fungus succumbed in about 6 months in wood that was allowed to dry rapidly in a shed that protected it from rain. Moderate freezing tempera-

TABLE 7.—*The effect of storage conditions on the survival of Ceratostomella ulmi in diseased native elm wood*

Storage treatment of wood sections	Survival period in months[a]
Nonparaffined	
Stored about 6 feet above the floor of a rain-proof tool shed in an open container	6
Stored in an open container and exposed alternately for one week at a time to −10° C. and then 15° C.	20
Stored at −3° C. in an open container	24
Paraffined	
Stored outdoors about 6 feet above ground level in an open porous container	20
Stored at −10° C. in an open container	22
Buried in the sand floor of an outdoor screen-covered cage to a depth of ½ the diameter of the sections	24
Stored in an open container and exposed alternately for one week at a time to −10° C. and then 15° C.	24

[a] The test continued for 24 months.

tures such as those that prevail at times during the winter in the New York City area did not appear to significantly shorten the survival period of the fungus. In fact, constant storage of diseased wood at temperatures of – 3°, and – 10° C. apparently helped to conserve the moisture in the wood necessary for the fungus.

Alternate freezing and thawing alone did not appear to have a pronounced deleterious effect on the organism. Under such conditions the organism lived longer in paraffined wood than it did in nonparaffined wood presumably because moisture was conserved in the former.

Competing organisms did not appear to materially affect the survival of *Ceratostomella ulmi*. In fact, it survived the 2-year period in wood that was partially buried in sand outdoors and this treatment was much more conducive to the development of competitive saprogens than any of the other treatments.

DISCUSSION

Ceratostomella ulmi grows best at moderately high temperatures. Despite this fact its growth activities are markedly slowed by temperatures above 30° C. and they practically cease at 33° C. or slightly above. While growth is possible over a rather wide range of temperature the organism is best adapted to that from 18° to 30° C. and this adaptation seems admirably suited to the saprogenic growth of the fungus in the areas of the United States where it prevails as a pathogen.

At temperatures below the range most favorable for mycelial growth, the organism can increase by budding. This phenomenon might help to intensify the Dutch elm disease in at least three ways: through an increase in the amount of inoculum at the source; through an increase of inoculum at the infection court; and through increased numbers of spores within the xylem vessels where they move freely to bring about rapid invasion of the tree (1, 5).

Moisture is a primary factor that may limit the saprogenic activities of *Ceratostomella ulmi*. Low air and substrate moisture may not only inhibit germination of spores but it may also suppress the growth of hyphae and the production of fruiting structures. Since the organism depends largely, for its continued existence and distribution, on the inoculum developed in pupal cells made by its principal insect vectors the suppression of this growth activity may well be critical. The degree to which the moisture factor is felt by the organism depends greatly upon its location and state of association with elm material. For example, in the form of spores and/or hyphae on the outer surfaces of elm wood the organism is unable to grow if the relative humidity of the surrounding air is less than 98 per cent. However, it can grow out from wood in which it became established in the form of hyphae during the disease process even if such material is kept in an atmosphere whose relative humidity is as low as 92 per cent. Thus, it appears that the latter more vital type of association enables the organism to obtain some water from the surrounding wood even when the diseased wood is exposed in relatively dry air.

Once the Dutch elm disease fungus becomes established in elm wood it is capable of persisting as a saprogen for rather extended periods under a variety of conditions. From the disease control viewpoint this is unfortunate because it means that dead, dying, and decadent elm material of all sorts that has not been debarked may constitute a source of inoculum. However, the inability of the organism to withstand rapid and prolonged drying, even when embedded in wood, is a characteristic that renders it vulnerable. Perhaps more concerted effort to make use of this fact in connection with sanitation work would be a forward step toward more satisfactory control of the Dutch elm disease.

SUMMARY

The optimum temperature for the germination of coremiospores of 2 cultural races of *Ceratostomella ulmi* was about 27° C. At 18° C. and lower the spores tended to bud. Other factors such as racial differences, age of spores, and nutrition affected spore germination.

The temperature range generally favorable for growth of the mycelium on potato-dextrose agar was 21° to 30° C. For the cultural races studied, the minimum temperature for growth lay between 1° and about 3° C., the optimum temperature was about 27° C., and the maximum was between 33° and 36° C.

The optimum temperature for development of coremia was about 24° C. Some cultural races formed coremia on elm wood at temperatures that ranged from 3° to 30° C., others did not form them below 9° C., while two races did not produce coremia at any temperature tried.

Coremiospores of *Ceratostomella ulmi* germinated well on glass microslides and on elm wood at relative humidities of 98 per cent and higher; they did not germinate at 96 per cent and lower.

Vitally established in the form of mycelium and spores in diseased elm wood the organism grew out over the surface at relative air humidities of 92 per cent and higher. Growth was very sparse at 92 per cent, fair at 96 per cent, and abundant at 98 and 100 per cent relative humidity.

Ceratostomella ulmi lived at least 2 years in diseased elm wood that was protected from rapid loss of water and from abnormally high temperatures. Conditions that induced and permitted rapid loss of water from diseased wood were generally unfavorable for the survival of the pathogen in such material.

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ANTHRACNOSE RESISTANCE IN FLAX[1]

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(Accepted for publication April 10, 1945)

Anthracnose of flax, caused by *Colletotrichum lini* (1, 3, 4, 5, 6, 7), is distributed throughout the flax-growing regions of the world. The disease first caused an appreciable loss in California flax in the season of 1940–1941 (5, 6). Houston (6) has estimated losses as high as 35 per cent in some fields during that season. Although recent losses have not been as marked, the organism is still present and could, when the season is favorable, again become destructive, especially since the flax variety still predominant in California, Punjab, is very susceptible to attack. In the North Central States, although flax anthracnose has acted as a seedling blight and canker and has considerably damaged developing plants in local areas in Minnesota, it has not been regarded as being especially serious (3).

Little has been published concerning the reaction of current flax varieties to *Colletotrichum*. The susceptibility of Punjab and the partial resistance of Argentine (C.I. 463) have been known for several years. More recently Houston reported (6) that Rio (C.I. 280) and the plant introductions C.I. 1008 and C.I. 1009 also have partial resistance. He reported that C.I. 1008 and C.I. 1009 carry a very high degree of resistance.

This paper records the anthracnose reactions of a number of flax varieties.

MATERIALS AND METHODS

The inoculations were on plants grown in 5-inch pots in the greenhouse from November, 1943, to March, 1944. Supplementary tests have been made from November, 1944, to February, 1945. The seed were planted in steam-sterilized Toxaway silt loam. The seedlings were thinned to 15 per pot. Duplicate pots were tested when the seedlings were 5–8 inches tall.

The culture of *Colletotrichum lini* was furnished by Dr. B. R. Houston,[3] to whom we extend our thanks. The culture has been increased on potato-dextrose agar.

Vigorously growing cultures of the fungus, about 10 days old, were used as a source of spores; and enough spores were scraped from the agar slants into distilled water to make the water turbid. The stems and leaves of the seedlings were sprayed (4, 7) by means of a small atomizer. A small air pump connected to the atomizer aids in securing a heavy, uniform inoculation. The susceptible Punjab variety of flax was included in each group of inoculated plants as a check upon the effectiveness of inoculum. After inoculation, the seedlings were immediately placed in a moist chamber and

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left for about 48 hours (Fig. 1) after which they were returned to the bench in a moist greenhouse. Observations were made 12–15 days after spraying at which time the symptoms were usually well developed.

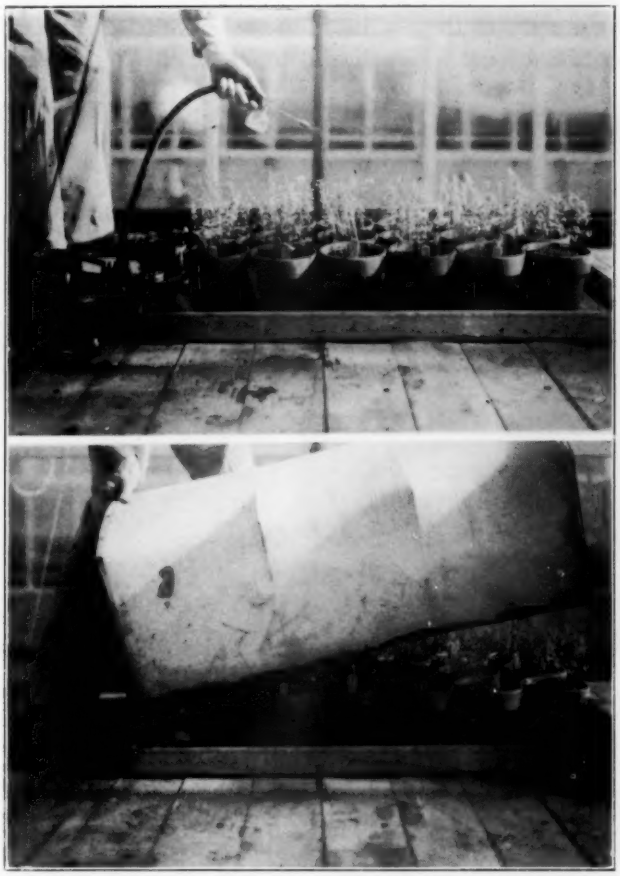

FIG. 1. Flax seedlings 5–8 inches tall are sprayed with a water suspension of spores of *Colletotrichum lini*. A small air pump aids in securing a heavy, uniform inoculation. After spraying, the inoculated seedlings are kept for about 48 hours under a cloth-covered frame which is kept moist.

RESULTS

The anthracnose on inoculated varieties ranged from severe infection of hypocotyls, stems, and leaves with much plant killing to very few small lesions on but few of the leaves (Fig. 2). No variety yet examined is regarded as being completely immune. For convenience, the varieties were placed in one of four groups according to the degree of infection: Group I—those with few, small leaf lesions but with no stem lesions and no plant killing (Fig. 2, D); Group II—those with a moderate number of somewhat larger leaf lesions and with some but not extreme leaf killing and with no stem lesions (Fig. 2, C); Group III—severe leaf infection with many leaves

attacked and large, rapidly spreading lesions causing much leaf killing. Stem lesions were present in varying amount (Fig. 2, B). Group IV—those with severe leaf and stem lesions. The stem lesions usually girdled the stem and killed the plant (Fig. 2, A).

Table 1 summarizes the results of the inoculations with *Colletotrichum*. The samples tested have been assigned to one of the four groups.

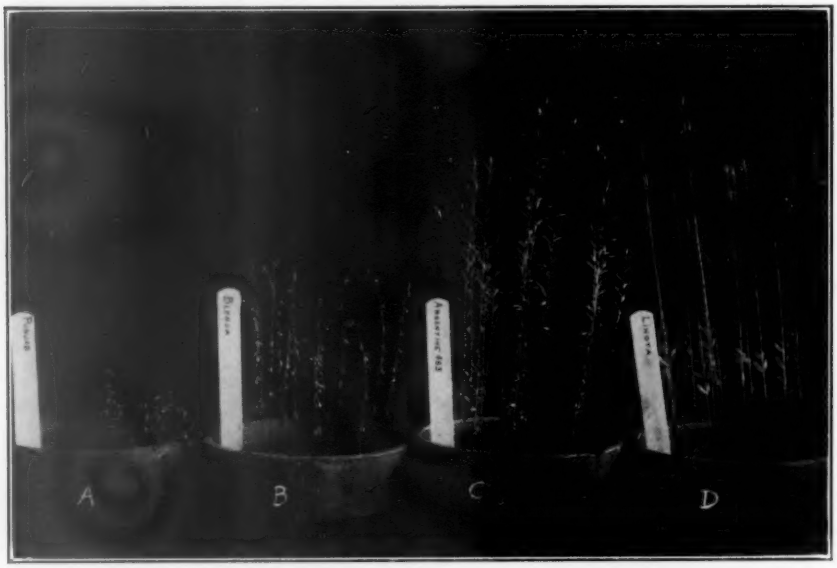

FIG. 2. Punjab, Blenda, Argentine 463, and Linota flax 14 days after inoculation with *Colletotrichum lini*. A. Very susceptible (Group IV). B. Susceptible (Group III). C. Moderately resistant (Group II). D. Resistant (Group I).

DISCUSSION

Table 1 records the amount of injury observed after spraying seedlings with a water suspension of spores of *Colletotrichum lini* and demonstrates that in these seedling tests the varieties differed in the amount of disease appearing. The results have been used to classify the varieties tentatively as resistant or susceptible as follows: Group I—resistant; Group II—moderately resistant; Group III—susceptible; Group IV—very susceptible.

It is believed that this classification according to resistance should not be too rigidly applied, especially for the intermediate classes. There are several reasons for caution. Primarily, our information on environmental effects is meager. Little precise information is available concerning the effect of soil or air temperature, moisture, light, or soil amendments on the virulence of the disease. Further, it is not recorded to what extent physiological races of the organism exist. The classification of some varieties is difficult, especially when considering the amount of leaf spotting. Different varieties grade from very susceptible to very resistant.

Greenhouse disease studies usually provoke the question of the applica-

TABLE 1.—*Amount of anthracnose injury 12–15 days after spraying flax seedlings with a water suspension of spores of Colletotrichum lini*

Variety	C.I.[a] No.	P.R.[b] No.	Class reaction[c]
Argentine	472	71–1	I
do	472	71–2	I
do	462	72–2	I
Buda	326	131–1	I
Buda 80	873	132–1	I
Crystal	982	22–1	I
do	982	22–2	I
Golden × Rio	980	30–3	I
Linota	244	82–1	I
Malabrigo	696	104–1	I
do	696	104–3	I
N. D. 40046	492	84–1	I
Portuguese Sel.	1021	198	I
Rio	280	14–1	I
unnamed	1008	718–1	I
do	1009	719–1	I
do	1009	719–2	I
Bison	389	3–1	I–II[d]
Argentine	463	73–2	II
do	463	73–4	II
do	463	73–22	II
Goar Sel. No. 5	1112	750–1	II
Cascade	.....	794	II
Blenda Line	.....	367–21	II–III[d]
Common Pink	479	777	II–III[d]
Fleischman	.....	445–21	II–III[d]
Giza	378	83–2	II–III[d]
Abyssinian	36	68–21	III
do	300	69	III
Ac. 179	.....	793	III
Blenda Line	.....	367–1	III
do	.....	367–2	III
do	.....	367–3	III
B. Golden Sel.	976	33–2	III
do	976	33–4	III
do	977	34–1	III
do	977	34–2	III
Cirrus	727	411–1	III
Hercules	.....	332–4	III
do	.....	332–21	III
J. W. S.	800	146–1	III
J. W. S. × Bison	1043	29–1	III
Liral 12	.....	487–1	III
Martin	1062	333–31	III
Morocco	376	124–1	III
Newland	188	138–1	III
Pinnacle	693	190–2	III
do	693	190–4	III
Redwing	320	12–1	III
Royal	828	16	III
Russian fiber	.....	478–4	III
Talmune	687–1	41–1	III
do	687–1	41–21	III
Texala	.....	485–4	III
Victory	1045	2–1	III
do	1045	2–2	III
do	1045	2–3	III
Viking	981	10	III
No. 98701	.....	796	III
No. 99407	.....	795	III

TABLE 1.—(*Continued*)

Variety	C.I.[a] No.	P.R.[b] No.	Class reaction[c]
Black Mountain	.....	88	III–IV[d]
B. Golden Sel.	976	33–1	III–IV[d]
Stamm 8	1074	355–1	III–IV[d]
Abyssinian	36	68–1	IV
do	302	70–1	IV
Albidum Type 12	.....	59	IV
Bold	802	54	IV
Bridgemohan Ganj	793	66	IV
Buxar	797	64	IV
Concurrent	.....	330–2	IV
do	.....	330–21	IV
Dutch Hybrid	.....	376–1	IV
Giza	378	83–11	IV
Gossamer	.....	371–4	IV
Hoshangabad	40	126	IV
Indian Type 2	.....	61	IV
do 4	.....	60	IV
do Sel. 6	.....	58	IV
do 121	.....	143	IV
Ismailpur	796	57	IV
Pinnacle	693	190–22	IV
Punjab	20	48–1	IV
do	20	49–2	IV
do	20	49–7	IV
Rafigang	798	56	IV
Sitamarhi	792	67	IV
Stamm 8	1074	322–1	IV
Talmune	687–1	41–2	IV

[a] C.I. refers to accession number of the Division of Cereal Crops and Diseases, U. S. Dept. Agr.

[b] P.R. refers to accession number of the Plant Research Department, California Central Fibre Corp. Numbers following dash indicate selected strains.

[c] Refers to Group number described in text and illustrated in figure 2.

[d] Somewhat intermediate between the two groups.

bility of the indoor observation to field conditions. There are, however, numerous instances in small grain work where the agreement between field and greenhouse disease observation is close. There are only a few recorded observations of the field reaction of flax to *Colletotrichum lini* which can be compared to greenhouse studies. The high susceptibility of Punjab flax to *Colletotrichum* in the field in California is well known and conforms to our greenhouse observation. A number of Indian types of flax were badly injured by *Colletotrichum* in our experimental field plots in 1942; these types were very susceptible in our greenhouse tests. Flor (2) records some observations on *Colletotrichum* in the field during 1944. He recorded the presence of leaf lesions, apparently caused by *Colletotrichum lini*, on Crystal flax near Morris, Minn., but stated that the anthracnose was not in epidemic form, and that it was not clear whether the poor stands were due to frost, weak germination, or seedling blight. Our greenhouse tests showed Crystal to have but few small leaf lesions after inoculation with *Colletotrichum*. Flor also records somewhat more abundant lesions of *C. lini* on Royal flax at Morris, Minn., and in a field of Victory near Grafton, North Dakota. Our greenhouse results rate both Royal flax and Victory flax as somewhat

more susceptible than Crystal. The greenhouse reaction of flax to *C. lini* is thus not inconsistent with the few field observations at hand. More data, however, are desired to throw more light on this question of field *vs.* greenhouse observations.

Nevertheless, the observations recorded in table 1 demonstrate that there are marked genetic differences between flax varieties in the amount of resistance to *Colletotrichum lini*. During the tests, Punjab flax seedlings were nearly always included as a check upon the severity of the inoculation. Under the conditions of these tests, the inoculation was always heavy enough to kill the Punjab seedlings.

Several generalizations are drawn from the data presented. None of the Indian varieties tested—Albidum, Bold, Bridgemohan Ganj, Buxar, Hoshangabad, Indian Types 2, 4, 6, 121, Ismailpur, Punjab, Rafigang, and Sitimarhi—have had any resistance to *Colletotrichum*. Various samples of Punjab have been tested repeatedly as checks, yet none has had any resistant plants. This would indicate that so far as anthracnose resistance is concerned no profit can be derived from individual plant selections of Punjab and, if a Punjab type is best in the California area, then the most likely approach would be through use of hybridization.

The Argentine group of flax varieties current among breeders in the United States carry some resistance to anthracnose.

As a group, the fiber flax varieties included in our tests seem to have little anthracnose resistance, though a few were classified in the moderately resistant Group II. None were classified as Group I. This result is contrary to the report of Hiura (4) who came to the conclusion that the broad-leaved varieties of seed flax are very susceptible to anthracnose, while the narrow-leaved varieties of fiber flax are comparatively resistant.

The existence of varieties carrying resistance to anthracnose—even under our extreme tests—encourages the conclusion that improved varieties can be obtained by hybridization. The large-seeded Argentine types, the taller seed varieties such as Linota, Bison, and Buda, the unnamed introductions C.I. 1008 and C.I. 1009, and others furnish a range of types of initial breeding material. Our results in breeding, to be published in a later paper, indicate that resistance is not too difficult to transfer. The results in general confirm the grouping in table 1. Crosses of the susceptible Punjab with the more resistant varieties, Argentine 462, Argentine 463, Argentine 472, Rio, Buda 80, Bison, Malabrigo, Linota, C.I. 1008, and C.I. 1009, segregated for resistance and susceptibility in the F_2; resistant and susceptible lines have been found in the F_3. Crosses of the moderately resistant Argentine 463 with susceptible Gossamer, Pinnacle, Martin, Talmune, and Cirrus also segregated in the F_2. Crosses of Punjab with susceptible Dutch Hybrid, Pinnacle, Cirrus, and Abyssinian gave a completely susceptible F_2. Crosses of resistant Argentine with resistant C.I. 1008 and C.I. 1009 did not throw any susceptible plants in the F_2. C.I. 1008, C.I. 1009, Linota, and Buda 80 appeared especially good in the variety tests for anthracnose resistance and have given good segregates in hybrids.

SUMMARY

The method used for testing seedlings of flax with *Colletotrichum lini* is described and the varieties are classified according to the amount of injury following inoculation. A range of types carrying anthracnose resistance is available for breeding material. C.I. 1008, C.I. 1009, Linota, and Buda 80 were especially free from anthracnose. Punjab in repeated tests has never had any resistant plants. Argentine types carry various degrees of resistance.

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A GRAFT-TRANSMISSIBLE VIRUS OF SWEET POTATO

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(Accepted for publication April 15, 1945)

While examining sweet-potato plots at Beltsville, Md., during the summer of 1942, a plant was found with symptoms indicating a virus infection. This plant was one of a variety designated as No. 029878 that had been propagated from roots received from Leningrad, U.S.S.R., in 1934. No other plants of this variety showed any evidence of the disease in 1942 and none had been noted in previous years. An examination of the remainder of the planting failed to show any evidence of similar infection in the many other varieties included in the trials. Cuttings were made from the supposedly mosaic plant and were rooted in the greenhouse, where a stock has since been vegetatively propagated for observation and experimental use.

SYMPTOMS

The symptoms on the original plant were typical of those associated with viruses of the mosaic (*Marmor*) group. These same symptoms have appeared consistently in artificially infected plants during two years' observations in the greenhouse. In many plants the primary symptom is a pronounced yellowing along the veins of the younger leaves. This yellowing may outline many of the very small veins in a manner suggestive of frost patterns on a window pane (Fig. 1, A), or may affect only certain sections of the veins as shown in figure 1, B. On other plants, the first symptom may consist only of small, diffuse, yellow spots from 1 to 2 mm. in diameter (Fig. 1, C). These spots, however, frequently occur together with some vein-clearing of the type mentioned. Symptoms of both types also are found on leaves produced after the plant has been infected for some time.

In later stages of the disease, the yellowing of the veins is often followed by the development of pale green areas that may occur as comparatively small spots or may be so large as to include one-third to one-half of the leaf (Fig. 1, D). In such leaves the darker portions often are abnormally dark green and may show small, feathery areas of yellow along the veins. The leaves are sometimes slightly rugose and dwarfed but not much distorted. Infected plants commonly have shortened internodes, and there is a definite stunting of the plant in late stages of the disease (Fig. 2). No necrotic symptoms have ever been noted on leaves, stems, or roots. The fleshy roots are smaller than those of healthy plants but infected plants in the greenhouse always produce a fair number of medium-size storage roots.

When mosaic plants grow slowly, the leaf symptoms are likely to be less pronounced than in rapidly growing plants. When growth is slow, there

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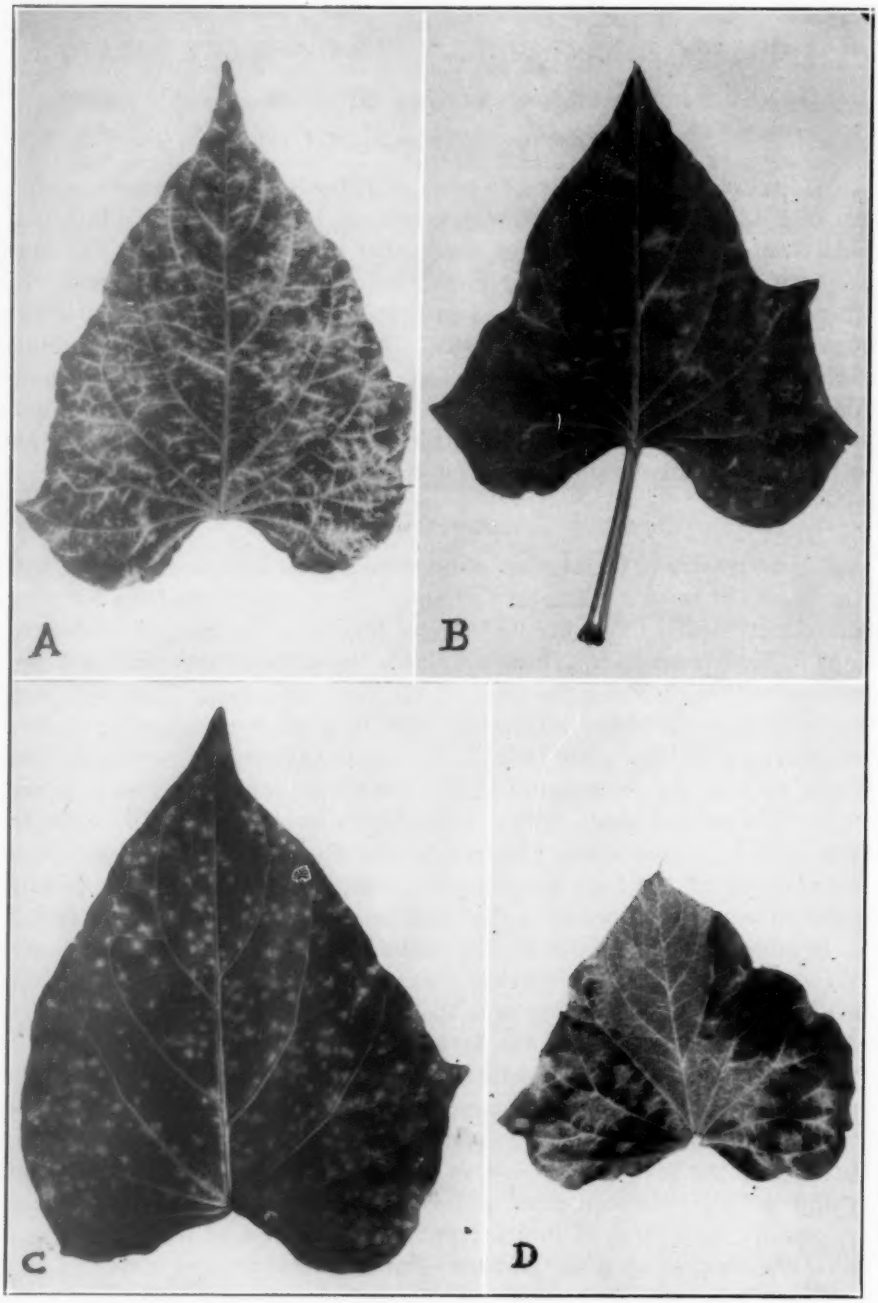

FIG. 1. Sweet-potato leaves with symptoms of mosaic. A, B, C.—Symptoms occurring in early stages of the disease and appearing to some extent on later growth. D.—Leaf with large light and dark green areas produced some time after infection.

usually are some leaves with only faint evidence of the disease but there is no general masking or extreme mildness of symptoms in the leaves of such plants. When plants are grown in the greenhouse at summer temperatures ranging from 75° to 105° F., the mottling is fairly pronounced. Temperatures as low as 60° to 65° F. do not suppress mosaic symptoms in the new growth.

The occurrence of mosaic symptoms on sweet-potato plants has been reported by various observers, chiefly in the Southern States. In many of these reports the symptoms were not described and, in all instances where descriptions are given, the symptoms of the disease appear to have been quite different from those produced by the virus with which we have been working.

Rosen (5, 6) reported a mosaic disease of sweet potato in Arkansas that appeared to be confined to the variety Nancy Hall. The leaves of diseased

FIG. 2. Nancy Hall sweet-potato plant (at right) showing stunting characteristic of late stages of mosaic infection. Inoculated by root-plug method. Healthy control (at left) grown from uninoculated half of same root.

plants commonly showed a marked narrowing of the blades and an irregularity of the margins, accompanied by a pronounced puckering of the leaf. The affected leaves also showed a mottling consisting either of very small, light-colored dots, streaks, and blotches generally distributed over the leaf, or of larger, irregular blotches of light green occurring between areas of normal color. The leaf symptoms of these plants, as shown in the colored plate and text figures of the two papers, are unlike the symptoms of the disease we have studied at Beltsville. Rosen reported (6) that the virus found in Arkansas was transmissible by rubbing the leaves with extracted juice of mosaic plants and by injecting the mosaic juices into the stem. No evidence of infection was obtained, however, except in a few instances where the inoculated plants were kept in continuous growth for 12 to 13 months. The plants were inoculated in the field during the spring and carried over winter in the greenhouse by means of cuttings that were again set in the field during the following spring. When the fleshy roots of inoculated plants were used for propagation, after storage during the winter, no evidence of the disease occurred during as many as three successive seasons. Stem

grafts and contact of cut surfaces of diseased and healthy roots did not produce infection in healthy plants.

Weber (7) described a mosaic disease, in Florida, that caused a stunting and rosette-like appearance of the affected plants. The larger veins of the leaves were wide and flattened in a way suggestive of fasciation. The leaf blades were narrow and filiform, with pouch-like protuberances between the veins and a crinkling of the leaf margins. There was little evident mottling of the leaves, but in other respects the symptoms appear to have been much like those described by Rosen although of a more severe type. No attempts were made to infect healthy plants by grafting but mechanical inoculations with the expressed juice of diseased vines were not successful. In a later paper (8) Weber and West reported what they considered to be a second type of mosaic on sweet potato but made no mention of any attempts to transmit a virus from diseased to healthy plants. The symptoms of this second disease included a yellowing of the veins which, in some respects, was not unlike that caused by the virus we have studied. However, there was a wrinkling and pouchiness of the leaf blades that we have never observed and the later mottling of the leaves appears to have been of a somewhat different type from that occurring in the infected plants of our experiments.

Weimer (9) studied a mosaic-like disease on the variety Nancy Hall but was unable to secure infection by mechanical inoculation with expressed juices of diseased plants. Grafting of diseased and healthy stems and the grafting of healthy shoots on diseased roots also failed to cause infection. He did not describe the symptoms of the diseased plants used as a source of inoculation but it is known that they were much like those reported by Rosen.

Harter and Whitney (3), working with the variety Nancy Hall, found that an air temperature of 38° C., either with or without high humidity, would cause complete masking of symptoms in plants affected with a disease much like that described by Rosen and apparently identical with the one studied by Weimer.

Wellman (10) reported that he was able to transmit the southern celery strain of ordinary cucumber mosaic to sweet potato by means of aphids and could recover the virus by the same means. Mechanical inoculations gave only negative results. The first symptoms consisted of a vein-clearing that appeared about 7 days after inoculation. This clearing of the veins later disappeared and was followed by a mild but definite mottling that became almost undetectable in plants approaching maturity. He stated that this disease was found on sweet potatoes in the field in Florida and Cuba. Wellman showed no photographs of symptoms but the disappearance of the vein-clearing and the eventual fading of the mottling of the foliage are unlike the symptoms of the disease that we have studied. The incubation period of the celery virus on sweet potato was also much shorter than any observed in our inoculation experiments.

In a recent report, Hansford (2) states that surveys made in 1944 show that sweet potatoes in certain sections of East Africa frequently are affected

with what appears to be a virus disease. The symptoms, which vary with the variety affected, include mottling, vein-banding, distortion or crinkling of the leaves, and some stunting of the plants, especially those of narrow-leaved varieties. Preliminary surveys indicate that the disease is of considerable importance. Nothing is yet known as to methods of transmitting the suspected virus but the spread of the disease in the field indicates the presence of an insect vector. The symptoms described by Hansford are suggestive of those of the disease found at Beltsville but no definite comparison can be made at this time.

TRANSMISSION OF THE VIRUS

Inoculations with Juices of Mosaic Plants

All attempts to secure infection by inoculation with expressed juices or crushed tissues of mosaic plants have been unsuccessful. A total of 103 plants were inoculated by rubbing the leaves with the expressed juices of mosaic leaves. Leaves of various ages were used as a source of inoculum and all inoculated leaves were dusted with carborundum powder before rubbing. Similar leaf inoculations were also made with the juices of stems, and of the feeding and the storage roots of mosaic plants.

Stem inoculations were made (a) by introducing expressed juices of mosaic stems or leaves into the stem by needle pricks or by injection with a hypodermic needle, (b) by the insertion of freshly macerated tissue of stems or leaves into longitudinal incisions in the stem, and (c) by the hypodermic injection of juices extruded from the ends of freshly cut stems of mosaic plants. A total of 92 plants were inoculated by one or another of these methods but none developed symptoms of mosaic.

All of the plants inoculated were held for periods of from 6 weeks to 8 months at greenhouse temperatures between 75° and 95° F. Since plants infected by grafting show symptoms within 14 to 28 days after the graft is made, it appears that infection is not readily obtainable by inoculations with expressed juices.

Transmission by Stem Grafting

Attempts at transmission of the virus by approach-grafting of stems of mosaic and healthy plants have been uniformly successful where union of the stems has occurred. The healthy and mosaic stems were joined by tongue grafts made about 4 to 6 inches back of the growing tip, the graft being wrapped with raffia and covered with moist cotton. Twenty of 22 such grafts resulted in the development of typical symptoms in the healthy plant, the remaining two grafts failing to unite. In most plants infected in this manner, the first symptoms appeared in the stem to which the mosaic runner was united. Occasionally, however, the first sign of the disease appeared in a runner other than that grafted, indicating a rather rapid passages of the virus down the stem. The incubation period of the virus varied from 14 to 28 days, with most of the plants showing definite symptoms within

20 days. The first symptoms consisted either of the development of small yellow spots on leaves which had not reached full size (Fig. 1, C), or there was a clearing of small segments of the veins similar to that shown in figure 1, B. All the runners of an infected plant eventually were diseased and the later symptoms included all the types shown in figure 1. Ten of the fleshy roots of the infected plants were grown in sterilized soil in the greenhouse and all produced shoots with typical mosaic symptoms.

Attempts were also made to secure infection by cleft-grafting a scion, taken from the tip of a mosaic shoot, on a healthy stem from which 2 inches of the tip had been removed. All of 20 such grafts failed to unite, but in 2 of the healthy plants evidence of infection appeared within 16 days after the graft was made. Since the mosaic scions in these instances had wilted and died within 24 hours, it would seem that the virus must have gained entrance into some tissue of the healthy stem, possibly the phloem, in which it was able to multiply with some rapidity. Attempts have since been made to secure infection by hypodermic injections and by needle pricks made at various points in the ends of freshly cut stems, but so far all have been unsuccessful. Wedges of freshly cut stem tissue inserted in longitudinal slits in the stem also have failed to cause infection.

Transmission by Insertion of Plugs of Mosaic Root Tissues in Healthy Roots

The simplest method of transmitting the virus to sweet potato has been the insertion of plugs from mosaic-diseased roots into roots of healthy plants by the method used by Goss (1) in work with viruses affecting potato [*Solanum tuberosum* L.]. The healthy sweet potato root was first cut in halves and one portion planted as a control. A core of diseased root tissue was then removed from the fleshy root of a mosaic plant with a cork borer and inserted in a hole made in the remaining half of the healthy root with a borer one size smaller. In many of these experiments actual union occurred between the mosaic root plug and the surrounding healthy tissue but, even when the plug eventually decayed, the shoots from the inoculated roots always developed typical mosaic symptoms. A total of 73 roots were inoculated in this manner and all developed mosaic shoots that did not arise from the cores of mosaic tissue inserted in the roots. All control roots produced only healthy shoots.

Tests for Transmission by Successive Cutting of Diseased and Healthy Roots

All attempts to transmit the virus by successive cutting of diseased and healthy roots have failed. In these experiments the healthy roots were cut in two with a sterilized knife and one portion planted as a control. The other portion was again sliced with a knife that was thoroughly coated with the juices of a freshly cut mosaic root. Thirty-four roots of either Nancy Hall or No. 029878 were treated in this manner but all the shoots from control and inoculated roots remained healthy.

Tests for Transmission Through Fibrous Roots

Soil in which mosaic plants were growing in the greenhouse was removed from the pots and passed through a one-half-inch mesh screen to remove all the larger fibrous roots. Two days later the soil was placed in sterilized 6-inch pots and one-half of a healthy root (Nancy Hall) planted in each pot. The remaining half-roots were planted in pots of steam-sterilized soil to serve as controls. Thirty plants were grown from roots planted in the contaminated soil but no infection was evident during the 90 days the plants were held in the greenhouse. Roots planted in sterilized soil produced only healthy shoots. Thirty healthy roots also were planted in unscreened soil that had grown mosaic plants and then had been stored for 30 days in a moist state in the greenhouse. All of the shoots of the plants of this series remained healthy.

Attempts to infect healthy plants by mechanical inoculation of the feeding roots also have been unsuccessful. The inoculations were made with juices of leaves, feeding roots, and storage roots but no infection occurred in the 24 plants thus inoculated.

Tests for Insect Transmission

No real attempt has been made to determine possible insect vectors of the virus but some observations indicate that it is not readily transmitted by aphids. During the summer of 1944, aphids appeared on runners of healthy and mosaic sweet potatoes growing in immediate contact in the greenhouse. No evidence of infection appeared in the healthy plants during 3 months' observation. Aphid-infested leaves from mosaic plants were removed and placed on leaves of 10 healthy plants, but here again there was no evidence of infection in plants held for 60 days. The aphids, from a superficial inspection, appeared to be *Myzus persicae* Sulz., which was present on peppers in the same house at the time.

Tests for Transmission by Dodder

Attempts have been made to transmit the sweet-potato virus by means of two species of dodder, *Cuscuta arvensis* Beyrich, and *Cuscuta californica* Choisy. Up to the present, however, we have not been able to establish either of these species on the sweet potato, but the experiments are being continued.

CROSS-INOCULATION EXPERIMENTS

Efforts have been made to transmit the sweet-potato virus to the Samsum variety of Turkish tobacco (*Nicotiana tabacum* L.), *N. glutinosa* L., *Datura stramonium* L., cucumber (*Cucumis sativus* L.), and morning glory (*Ipomea purpurea* Lam.). All of these plants were inoculated with the expressed juices of mosaic leaves of sweet potato, using the leaf-rubbing method with carborundum powder. The number of plants inoculated varied from 9 to 30. No infection was obtained in any series.

Attempts also have been made to infect sweet potato (Nancy Hall) with the viruses of ordinary tobacco mosaic and the southern celery strain of ordinary cucumber mosaic. Thirty plants have been inoculated with each of these viruses by the leaf-rubbing method, using carborundum powder. No infection was obtained from any of these inoculations.

VARIETAL SUSCEPTIBILITY

No extensive tests of varietal susceptibility have been made, but all the varieties tested have proven susceptible to the virus. These inoculations have been made by the root-plug method, using from 3 to 9 plants of each variety. Infection occurred in all the inoculated plants, the controls all remaining healthy.

The named varieties infected were: Nancy Hall, Porto Rico, Triumph, and Wennop. Varieties received from the Division of Plant Exploration and Introduction under the numbers 129651 and 129653 also were infected, as were seedlings listed as B-219, B-2496, and B-2521. All of these plants showed symptoms of the type described and all appeared to be equally severely affected.

DISCUSSION

Apparently the mosaic disease of sweet potato found at Beltsville is not common in the field. The symptoms are generally so striking that diseased plants are readily noted and, if it were of frequent occurrence, one would expect more reports of it. We have not attempted to determine the distribution of the disease, however, and while the files of the Division of Mycology and Plant Disease Survey contain a number of reports of the occurrence of "mosaic" in various States, it is not possible to say how many of these may have referred to the disease we have studied.

Since the virus is not readily transmissible except by tissue union or prolonged contact of diseased and healthy tissue, it is not likely to be widely disseminated by means other than an insect vector. So far, the lack of evidence regarding such vectors prevents any statement as to the likelihood of its general distribution.

It seems probable that this virus is distinctly different from those whose transmission or other behavior were studied by earlier workers. The symptoms of the mosaic diseases investigated by Rosen, Weber, Weimer, and Harter and Whitney all seem to have been much alike and were essentially different from those of the disease we have studied. The extremely long incubation period reported by Rosen is unlike that of our virus and if, as indicated by the results of Rosen and of Weimer, the causal agent was not transmitted by grafting, there would be much doubt as to whether a virus was actually concerned. It is possible, however, that further attempts with such methods might have produced infection in healthy plants. Wellman's description of the symptoms in sweet potato infected with the southern celery strain of the cucumber-mosaic virus does not correspond with what has been

observed in plants infected with our virus, and the short incubation period that he mentions is a further indication of a difference in the two viruses.

The so-called second type of mosaic described by Weber and West was characterized by a yellowing of the veins that is suggestive of that found in our infected plants. Although the later symptoms of this "second" type were rather different from those produced by our virus, it is not impossible that the two viruses were of a similar type. Since no comparative material is available, however, and since no attempts at transmission of the second virus were reported, it is not possible to make a definite statement as to the relationship of this virus to ours. The same situation exists with the virus reported by Hansford in East Africa although here again there is a possibility that the virus he describes is very similar to the one we have studied at Beltsville.

Since our virus appears to differ both in symptoms and methods of transmission from that described by Rosen under the name of sweet-potato mosaic, it is suggested that it be known as feathery mottle of sweet potato. Under the system of classification of the genera of plant viruses proposed by McKinney (4) it should be placed in the genus *Flavimacula* since it is transmitted only by tissue union or prolonged contact of diseased and healthy tissue, and insect vectors are unknown. It is proposed that it be known as *Flavimacula ipomeae* sp. nov. with the following description.

Flavimacula ipomeae sp. nov.

Common name of disease.—Feathery mottle of sweet potato.

Host reaction.—In *Ipomeae batatis* L. var. Nancy Hall, and other common varieties of sweet potato, first induces a finely branched yellow mottle along the small veins with or without the development of many small, diffusely margined spots 1 to 2 mm. in diameter. Leaves produced after infection slightly rugose but not much distorted; mottled with areas of light and dark green and showing feathery patches of yellow in dark portions of the leaf. No necrosis of leaves, stem, or roots. Plants considerably dwarfed. Not known on hosts other than sweet potato.

Transmission.—Limited to tissue union by approach grafting of stems or by insertion of cores of diseased root tissue in healthy roots. No insect vector known.

Physical and chemical properties.—Not known.

Distribution.—Observed only at one locality in Maryland.

SUMMARY

A virus disease of sweet potato causing an unusual type of feathery yellow mottling has been observed at one locality in Maryland. It does not appear to be common in the field. The infected plants are considerably dwarfed but there is no pronounced distortion of the leaves and no necrosis of stems, leaves, or roots.

The virus has been transmitted by grafting of diseased healthy stems and by the insertion of cylindrical plugs of diseased root tissue into healthy roots. No transmission has been secured by other methods of artificial inoculation and no insect vector is known.

The virus produces symptoms distinctly different from those mosaic diseases of sweet potato whose transmission was studied by earlier workers and it is proposed that it be known as feathery mottle of sweet potato.

Under the system of generic classification proposed by McKinney it is suggested that it be listed as **Flavimacula ipomeae** sp. nov.

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MOVEMENT OF MOSAIC VIRUS THROUGH SUGAR-CANE SEED PIECES

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(Accepted for publication April 17, 1945)

In Louisiana, sugar cane is propagated by planting seed pieces, these usually being whole stalks. On each stalk there are usually from 10 to 20 buds or eyes, each of which, if sound, is potentially capable of developing a shoot. In the field, however, even under the best of conditions, rarely more than 20 to 25 per cent of the buds produce shoots which survive. For these seed pieces, the term "seed cane" is ordinarily used.

Usually when a mosaic-diseased seed piece is planted, all the shoots growing from it have the mosaic symptoms. Conversely, mosaic-free seed pieces produce only mosaic-free shoots. To have available mosaic-free plants for inoculation purposes, mosaic-free seed pieces are planted either in the greenhouse or in isolated places in the field. The chance of such plants picking up the infection during the early stages of growth is very slight.

During the winter of 1941–42, while conducting some inoculation tests in the greenhouse on young shoots of two varieties, Co. 281 and C.P. 28/19, which had grown from mosaic-free seed pieces, it was observed that not only did the plants that were inoculated develop the mosaic symptoms, but mosaic developed in some other shoots close by which were not inoculated. When the latter shoots were removed from the soil it was found that they were attached to the same seed pieces as were the inoculated shoots. Since there was no evidence of what could have been interpreted as natural infection, *i.e.*, transmission by insects, in other cane growing in the greenhouse, the possibility of virus movement through the old seed piece was suspected. Following this, field inoculations were made to determine whether or not there may be a transfer of the virus through the seed piece, and, if so, what significance is to be accorded such a method of mosaic spread in sugar-cane fields. These tests were made during three growing seasons.

All tests were carried through in a similar way. Full-length stalks were planted and then a single shoot developing from each seed piece was inoculated by placing a few cc. of extracted juice from a mosaic-diseased plant in the whorl of leaves at the top of the plant, then pricking through the spindle a number of times with fine needles. When shoots from other nodes showed the mosaic symptoms, the seed pieces were dug and the presence or absence of mosaic was recorded for all shoots which had developed from the different nodes.

Young shoots developing from buds on the nodes sucker freely and consequently instead of a single plant there is soon a cluster of plants, commonly called a stool, growing from a node. Usually the mosaic will spread rapidly from one plant to the others in a stool. In the following discussion the term "shoot" is used indiscriminately for a single plant or one among the many in a stool.

Tests in 1942. Data were obtained from three inoculation experiments. Two varieties, C.P. 28/19 and C.P. 28/70 were used. A total of 50 plants were inoculated; 29 of them developed mosaic. Virus spread, apparently, from 10 of these inoculated plants to noninoculated plants.

From the standpoint of the presence or absence of mosaic, the condition of all the shoots on these seed pieces in which there appeared to be a movement of the virus is shown in table 1. It will be observed that the virus spread from the inoculated shoots in both directions in the seed pieces, but in some cases passed by certain nodes and infected shoots further removed from the ones inoculated. In figure 1 is shown one of the seed pieces (test A, seed-piece No. 9) at the time it was removed from the soil.

TABLE 1.—*Spread of virus in seed pieces, 1942; based on occurrence of mosaic in shoots on nodes other than the one at point of inoculation*

Test	Seed piece number	Nodes numbered from point of inoculation									
		Basipetal spread						Inoculated shoot	Apical spread		
		6	5	4	3	2	1		1	2	3
A	2	.....	+	0	.....	+	+	I	.....	.....	.....
	3	.....	.....	+	+	+	0	I	.....	.....	.....
	9	+	.....	+	.....	.....	+	I	0	.....	.....
B	2	.....	.....	.....	.....	+	.....	I	.....	.....	.....
	4	.....	.....	.....	+	.....	0	I	.....	.....	.....
	6	.....	.....	.....	.....	+	+	I	0	0	0
	18	+	.....	+	.....	+	.....	I	+	.....	.....
C	9	0	0	+	0	0	+	I	0	+	+
	17	.....	.....	.....	.....	.....	.....	I	.....	+	.....
	19	.....	.....	+	+	+	+	I	0	0	.....

Symbols: I = Inoculated shoot.
+ = Shoot with mosaic.
0 = Shoot with no mosaic symptoms.
On nodes without symbol, shoots not present.

Tests in 1943. Data were secured from four tests in 1943. The varieties C.P. 28/70 and Co. 281 were used. A total of 55 Co. 281 plants was inoculated. Mosaic developed in 17 of these. Virus spread from 6 inoculated plants to a total of 9 other shoots in 6 of the 17. With C.P. 28/70, a total of 45 plants was inoculated; 18 of them developed mosaic. Virus spread from 8 of the inoculated plants to a total of 14 noninoculated plants, apparently through the seed pieces.

Tests in 1944. In 1944, data were secured from 4 tests, 2 with the variety Co. 281 and two with C.P. 28/70. Two of the tests (A and B) were started in May and 2 (C and D) in June. As the results in general were very similar to those obtained in preceding years, they have been summarized in table 2. In all the tests mosaic was observed on some shoots that were not inoculated, apparently due to the transference of the virus through the old seed pieces. The virus seemed to travel in the seed piece equally well in either direction, some of the infected shoots being toward the base from the

inoculated one and others toward the top. In some cases, also as in previous years, the virus passed by some of the nodes without entering the shoots and infected others further along the stalk.

In table 2 the shoots under the heading "with mosaic" include the inoculated shoot. In all seed pieces, then, on which there was observed more than one shoot with mosaic, it is assumed that there was a transference of the virus from the inoculated shoot to the others. In test A, such a transference occurred in 14 out of 20 seed pieces; in test B, 13 out of 19; in test C, 6 out

FIG. 1. There were 9 buds (eyes) on the seed piece. Plants developed from buds 2, 4, 7, 8, 9. Plant 8 was inoculated. Mosaic developed in the inoculated plant and its suckers and also in plants from nodes 2, 4, and 7. Plants from node 9 remained mosaic-free.

of 21; in test D, 3 out of 25. Possibly the lower percentages in the two later tests can be explained by the greater deterioration in the seed pieces at the time the shoots were inoculated.

DISCUSSION

Sugar-cane mosaic is a virus disease known to be transmitted from plant to plant principally by insects. Infection is brought about naturally by certain insect vectors which suck the infectious juice from mosaic-diseased plants and then transmit it by feeding on healthy plants. In artificial inoculation experiments the commonest method of virus transfer is the injection of extracted juice from mosaic-diseased plants into the spindle of growing plants. Symptoms appear in the new growth usually within ten days to

three weeks. The rate of growth of the sugar-cane plants seems to determine time elapsing between inoculation and the appearance of the symptoms. Rapidly growing plants develop mosaic more quickly than those growing slowly.

In sugar-cane plots planted with mosaic-free seed cane and well-isolated from mosaic-diseased sugar-cane or corn, very little mosaic develops from natural infection, especially during the early growing season, which in

TABLE 2.—*Movement of mosaic virus through sugar-cane seed pieces. One plant per seed piece inoculated*

Seed piece number	Test A, C.P. 28/70		Test B, Co. 281		Test C, C.P. 28/70		Test D, Co. 281	
	Number of shoots		Number of shoots		Number of shoots		Number of shoots	
	With mosaic	Mosaic-free	With mosaic	Mosaic-free	With mosaic	Mosaic-free	With mosaic	Mosaic-free
1	3	1	2	0	3	3	1	1
2	3	1	.....	.....	1	1	2	1
3	4	1	3	0	1	1	2	1
4	1	1	1	1	1	3	2	1
5	2	0	3	0	1	1	1	2
6	3	0	1	0	2	2	1	2
7	2	0	4	1	1	3	1	2
8	2	1	3	0	1	0	1	0
9	3	0	1	1	1	2	1	1
10	4	1	1	1	3	0	1	0
11	3	0	2	1	2	0	1	1
12	7	0	1	1	1	0	1	1
13	2	1	2	1	2	0	1	0
14	0	3	3	0	1	0	1	0
15	1	5	1	1	1	0	1	1
16	4	0	2	2	1	0	1	1
17	1	1	3	0	1	0	1	0
18	1	0	2	0	1	1	1	1
19	1	0	2	0	3	2	1	0
20	5	1	3	0	1	0	1	0
21	.....	.....	.....	.....	1	1	1	0
22	.....	.....	.....	.....	.....	.....	1	0
23	.....	.....	.....	.....	.....	.....	1	2
24	.....	.....	.....	.....	.....	.....	1	0
25	.....	.....	.....	.....	.....	.....	1	1
Total	52	17	40	10	30	20	28	19

Louisiana extends into early June. Consequently, when it was observed that plants other than those inoculated developed mosaic, virus transfer through the seed piece was suspected. This suspicion was strengthened by the fact that no natural infection was found in the same varieties in other adjacent parts of the field where no artificial inoculations had been made. To confirm this, inoculation experiments were made in the field during three seasons. These were so designed as to give results that would answer the question as to whether sugar-cane mosaic can spread from plant to plant through the seed piece. The results seem to justify the conclusion that such a method of mosaic spread in sugar cane in Louisiana not only does occur but that it may occur quite commonly. The virus may travel from the point of inoculation either toward the top or toward the base of the seed piece.

Of particular interest, physiologically speaking, is the fact that the virus passes through the seed piece, skipping some plants but infecting others further removed from the point of inoculation. The virus apparently did not travel through all the seed pieces. This perhaps may be explained by the fact that portions of the stalks often disintegrate during the winter and spring seasons before or after the development of the young shoots. It is not probable that the virus would spread through dead or injured stalk tissues.

Whether under ordinary field conditions there is a transference of the virus through enough of the seed pieces to be of significance has not been determined. It is known, however, that mosaic spreads more rapidly in plant cane (plants from seed pieces) than in stubble cane (plants from rootstocks remaining in soil after harvest). There is a possibility that the greater spread in plant cane as compared to stubble cane may be due, in part at least, to the transfer of virus through the seed pieces from infected to noninfected plants.

SUMMARY

Sugar cane is propagated vegetatively by planting whole stalks or portions of stalks. Usually a number of shoots develop from each seed piece.

From inoculation experiments, it has been found that the mosaic virus can spread from an infected shoot through the old seed piece to other shoots. The virus spreads through the seed piece in either direction. The virus can also pass by a node and infect other shoots further removed from the point of infection.

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RHIZOCTONIA BUD ROT OF STRAWBERRY PLANTS

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(Accepted for publication May 12, 1945)

In 1935 Brooks[2] reported on a bud rot of strawberries in Florida and attributed it to a *Rhizoctonia* of the *solani* type. He described the disease as a "dry rot of leaf- and flower-buds, and bases of petioles and stipules, the last causing the older leaves to lose their upright position and lie flat on the ground." Brooks isolated the fungus and was able to reproduce the disease in the greenhouse.

What appeared to have been the same disease has been observed by the writer during the past few years in Arkansas, Delaware, Maryland, Mississippi, North Carolina, and Tennessee. Bain[3,4] reported observations on the disease in Louisiana and Mississippi in 1944.

This disease shows best in the field for a few weeks after winter dormancy has been broken and the plants have resumed growth of the central cluster of leaf and flower buds. In Florida this period occurs in December and January, in southern Louisiana and Mississippi in February and March, in Arkansas and Tennessee in March and April, and in Delaware and Maryland in April and May.

The first sign of the disease is an apparent retardation in growth of crown buds of infected plants as compared with normal ones. Later both leaf and flower buds turn brown and die, and are easily lifted out of their position. Simultaneously the outer previous year's leaves assume a more or less horizontal position and become darker green than leaves of normal plants. Several adventitious leaf buds soon develop under the residue of the originally killed buds and these may likewise be killed, or a few may survive but produce at first a weak, spindly growth, and later the usual form of "multiple crown" plants. Sometimes plants are killed outright. Usually, however, infected plants recover but produce no fruit that year. In dissecting the crown of an infected plant, one finds that buds have large, soft lesions or that they are partially or completely disintegrated. Another symptom of the disease, which is an after-effect of the early stage of bud disintegration, is that some leaves of surviving buds are injured before they unfold and after expanding are necrotic at the tip and margin of the leaflets. Sometimes an entire leaflet is missing. The effect of this injury persists throughout the life of the leaves. The dead leaf tissues slough off, leaving misshapen, puckered, or crinkled leaves. Brooks[2] has observed that stolons

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[2] Brooks, A. N. Rhizoctonia bud rot of strawberry. (Abstr.) Phytopath. **25**: 965. 1935.

[3] Bain, Douglas C. Strawberry diseases in Mississippi. U. S. Dept. Agr., Plant Dis. Reptr. **28**: 165. 1944.

[4] ———. Diseases of strawberries in Louisiana and Mississippi. U. S. Dept. Agr., Plant Dis. Reptr. **28**: 259. 1944.

and fruits are also attacked by the same fungus that causes bud decay. The stalks and root systems of strawberry plants are not attacked by the fungus, and this bud rot should not be confused with a Rhizoctonia root disease reported by Zeller[5] as causing extensive damage to strawberry roots in Oregon.

As a rule, the disease is of slight importance in strawberry fields, affecting only a few widely scattered plants. It is absent from many fields, but occasionally causes considerable damage. The most the writer has seen occurred in a single field near Van Buren, Ark., in which 25 or 30 per cent of the plants were estimated to be infected. Bain[3] observed the disease near Meridian, Mississippi, in 1944 and reported that in one field fully 30 per cent of the plants were infected. It is not known why the disease may become epiphytotic in some fields and be absent from others. It has been observed, however, that plants seem to be predisposed to infection when they are partially covered with wind-blown sand or soil, or when flood water has deposited some soil around the plants in low parts of a field.

The fungus is thought to be one of the variants of *Rhizoctonia solani*. When grown upon corn-meal agar it forms reddish-brown, loose, aerial hyphae with numerous black sclerotia. The sclerotia, in an early stage of development, are small aggregates of loosely constructed, short-celled, white hyphae, but later turn brown and become more compact. In the final stage they are globose to flat, dark brown to black, and from 0.5 to 1.25 mm. in diameter. They may form on the surface of the agar, on the inside wall of the culture tube, or among the aerial hyphae. The perfect stage of the fungus has not been seen on strawberry plants nor on artificial media.

Rhizoctonia bud rot may easily be mistaken for other strawberry diseases occurring during early spring. A. N. Brooks writes (correspondence), "It appears that the Rhizoctonia bud rot of strawberry can be confused with several things. Before I discovered the true cause I thought that it was the work of the sucking insect Pamera, which are numerous at times in Florida." The strawberry crown-rot disease, described by Plakidas[6] from Louisiana and caused by *Sclerotinia sclerotiorum*, is superficially similar to bud rot and not easily distinguished by field examination unless the black sclerotia of the *Sclerotinia* are found among the dead crown buds. Bain, in discussing the similarity of the two diseases, said, "This disease (Rhizoctonia bud rot) is very much like the Sclerotinia crown rot, but there is no damping off of basal leaves, nor are sclerotia present on or around the plants."

The spring dwarf disease, caused by the nematode *Aphelenchoides fragariae*, may also be mistaken for Rhizoctonia bud rot. Both diseases appear at the same time of year, and the symptoms are very similar during the initial stage, *i.e.*, both retard the new spring growth of leaves and flower development. The symptoms of the two diseases are more easily distinguishable at a later period. As a rule, the dwarf disease kills only the

[5] Zeller, S. M. A strawberry disease caused by *Rhizoctonia*. Oreg. Agr. Expt. Stat. Bull. 295. 1932.

[6] Plakidas, A. G. Factors responsible for the small strawberry crop this year. U. S. Dept. Agr., Plant Dis. Rptr. **19**: 132–133. 1935.

TABLE 1.—*Results of inoculating strawberry plants with Rhizoctonia*

No. plants	Treatment	Results
6	Uninoculated, crowns exposed	None infected
6	Inoculated, crowns exposed	All infected
6	Uninoculated, crowns covered with soil	None infected
6	Uninoculated, crowns covered with sand	None infected
6	Inoculated, crowns covered with sand	All infected

flower buds. The new leaves, however, are abnormal in appearance and development, because of the injury done to them by the great number of nematodes that have fed upon them during the early bud stage. The result of the nematode injury shows later as distorted, reddish leaves with short petioles and small, narrow leaflets.

Rhizoctonia attacks plants in no pattern of regularity, grouping, or segregation. There may be one or a few bud-rot-infected plants among a group consisting of an original set plant and its family of runner plants, or single bud-rot plants may be widely scattered. In contrast to the irregular distribution of Rhizoctonia bud rot, plants affected with nematode-dwarf disease are almost invariably grouped, because the nematodes spread from the infested mother plant to several or all of its daughter plants.

When this bud-rot disease was found in 1942 on the U. S. Plant Industry Station Farm at Beltsville, Maryland, work was initiated to determine the cause of the trouble. It was suspected that either *Sclerotinia* or *Rhizoctonia* would be found. Cultures were made and diseased plants were held in moist chambers at different temperatures. *Sclerotinia* never developed by any method tried, nor were sclerotia ever found associated with the disease in fields. A *Rhizoctonia* was isolated more frequently than any other one organism. In 1944 the pathogenicity of this fungus was determined in greenhouse experiments. Brooks' findings in 1935 were confirmed.

In a preliminary test started March 4, 1944, 30 plants were set in 6-inch pots filled with unsterilized composted soil. The plants were inoculated by

FIG. 1. The two end plants were inoculated with a culture of *Rhizoctonia*. The normal plant at center was not inoculated.

placing a small piece of agar containing hyphae and sclerotia of the fungus immediately under the surface of the soil and near the crown of the plants. Table 1 shows the arrangement of the test and the results.

The symptoms of infected plants were essentially identical with those of field-infected plants (Fig. 1). The crowns of a portion of the plants were covered with soil or sand, to determine if covering alone would cause abnormal growth of the plants similar to that caused by the *Rhizoctonia*. The covered plants developed slowly and irregularly, but the leaves finally emerged and plants were normal.

In April, 1944, a second greenhouse test was set up to demonstrate the association of the *Rhizoctonia* with strawberry bud rot. One hundred strawberry plants taken from the field were set in 6-inch pots so that the crown buds were slightly above the soil surface. Sixty-three of the plants were inoculated, the remainder were held as controls. At the termination of the test 30 days later, none of the 37 controls had any evidence of infection. Of the 63 that were inoculated, 40 had typical bud-rot symptoms.

This Rhizoctonia bud-rot disease of strawberries can and does occasionally cause a material loss to the berry crop in some fields. The motive for preparing this paper, however, was not to emphasize the economic importance of the disease, but principally to point out to those interested that there are other diseases of this crop occurring simultaneously and with almost identical symptoms that may easily be mistaken for bud rot. This paper also indicates the known distribution of the disease.

SUMMARY

A bud-rot disease of strawberries caused by a *Rhizoctonia* of the *solani* type, first reported by Brooks from Florida in 1935, was later found in Arkansas, Delaware, Maryland, Mississippi, North Carolina, and Tennessee.

The fungus attacks and kills the flower and leaf buds during a few weeks when buds resume new growth. This period embraces December and January in central Florida, February and March in southern Louisiana, and April and May in Maryland.

The early symptoms of the disease are very similar to and can easily be mistaken in the field for some other strawberry disorders, namely, injuries caused by the sucking insect *Orthea vincta* in Florida; the crown-rot disease caused by *Sclerotinia sclerotiorum* in southern Louisiana; and spring dwarf, caused by the nematode *Aphelenchoides fragariae*.

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FUNGICIDES IN RELATION TO SCAB AND FRUIT RUSSET OF PEAR IN THE HOOD RIVER VALLEY, OREGON[1]

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(Accepted for publication June 2, 1945)

Some pear varieties must be practically russet-free to command top prices on the market. This is particularly true for Anjou and Comice, two of the choice winter varieties grown in Oregon. While several conditions are known that cause smooth-skinned pears to become russeted, spray russet is one of the common causes for lowered cash returns from the crop.

Pear scab (*Venturia pyrina* Aderh.) has been in Oregon for many years, but it has been especially troublesome in the main pear-growing districts since 1932 (7). Lime-sulphur, and some of the other relatively efficient types of sulphur may give good commercial control of scab, but they may severely russet the fruits of some varieties and may injure any variety if followed by oil-containing sprays within 45 days. No really satisfactory fungicide has been available in the past that would adequately control scab without producing considerable russet on spray-sensitive pear varieties during most seasons. The present paper reports attempts during the past 12 years to find a satisfactory fungicide for use on Anjou pears in Oregon.

OBSERVATIONS ON RUSSET OF PEAR FRUITS

The origin of various types of fruit russet can sometimes be stated with certainty, but more often, the causes are difficult to determine, particularly in midseason or later.

Several conditions that are known to cause russet on Anjou pears when present at one stage or another in fruit development are: 1. Injuries due to insects, including blister mite, thrips, European red mite, and rust mites. 2. Mildew infection. 3. Environmental factors—excessive moisture and frost injury at various stages of development. 4. Inherent factors—bud sporting and phases of the black-end trouble of pears on Oriental rootstocks. 5. Mechanical injuries. 6. Spray injuries. Frost damage has affected unopened buds during 3 of the past 12 years. The frost russet injury, except when frost rings develop on the fruit, is difficult to distinguish from that caused by certain fungicides or other agents.

Many of the spray materials tested during the past 12 years have caused a definite russet on Anjou pears and had to be eliminated from further consideration. Spray injury is more likely to occur early in the season, the

[1] Published as Technical Paper No. 461 with the approval of the Director of the Oregon Agricultural Experiment Station and the Chief of the Bureau of Plant Industry, Soils and Agricultural Engineering, Division of Fruit and Vegetable Crops and Diseases, United States Department of Agriculture.

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sensitive period lasting until after the pubescence has been shed from the young fruits and the hair-attachment injuries have healed. This process is generally completed by the middle of June. After this it is possible to use with safety some of the sprays causing early injury, but it is then too late for efficient scab control.

EARLY SPRAY TESTS

Trees receiving no spray for the entire season often produced fruit with considerable russet injury. In many cases the russet appeared to result from infestations of blister mite and other insects that respond to control by dormant lime-sulphur applications. It was also discovered that the scab fungus overwintered as twig infections (8) which were important in causing primary infections some seasons. Since lime-sulphur was effective in inactivating the open scab lesions in early spring (8), check trees as well as other trees in a plot received a delayed dormant application of lime-sulphur or lime-sulphur-oil (2, see p. 6). This practice gave more uniform results in the evaluating of fungicides by eliminating some of the insect pests capable of causing a fruit russet, and by giving all trees in a plot a nearly equal chance for early scab infection. Lead arsenate at the regular 3 to 100 rate was combined with each test fungicide for the calyx application, and at least one additional application of lead arsenate was made later in the season for codling-moth control.

Spray materials tested for pear scab control may be conveniently classified into 3 general groups: 1. Those that caused definite russeting on Anjou pears, but otherwise might be efficient fungicides. 2. Materials that commonly cause little fruit injury, but gave poor scab control. 3. An intermediate group giving fair or good scab control without causing excessive fruit russet during most seasons. The fungicides falling in each group are listed below:

Group 1. Fungicides usually causing russet on Anjou pears: Basic copper sulphate, Bordeaux mixture in various combinations, copper-Bordo, copper oxide (red), copper oxalate, copper silicate, copper-hydro "40," copper oxychloride, copper acetonate, lime-sulphur in various combinations, dry lime-sulphur, ammonium polysulphide, flotation sulphurs, colloidal sulphurs, gas-house sulphurs, and micronized wettable sulphurs.

Group 2. Fungicides giving poor scab control: Copper oxide (black), phenothiazine, zinc-Bordo, zinc sulphate, 66A, fused bentonite sulphurs, and wettable sulphurs coarser than 325-mesh.

Group 3. Fungicides in general use or having commercial promise for scab control on Anjou pears: Wettable sulphurs of approximately 325-mesh, copper phosphate mixture, and ferric dimethyldithiocarbamate (Fermate).

Only copper phosphate mixture, of the fungicides tested up to 1941, compared favorably with the wettable sulphurs, which have been standard for scab control on Anjou pears. The copper phosphate-lime-bentonite mixture (3) was used at a 4–8–4–100 ratio in earlier tests, and later at 4–4–4–100 to reduce some of its bulk. The wettable sulphurs, with the exception noted

TABLE 1.—*Scab and russet injury*[a] *on Anjou pears in a single orchard for 7 years*

Year	Cover sprays	Check trees		Copper phosphate		Wettable sulphur[b]	
		Scab	Russet	Scab	Russet	Scab	Russet
	Number	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*
1935	4	30.4	1.2	2.4	2.0	4.1	2.5
1936	3	9.0	6.9	0.8	3.3	0.8	20.1
1938	4	32.5	18.9	1.3	13.8	1.9	86.0
1939	2	0.3	50.0	0.0	26.0	0.2	51.2
1942	4	56.4	5.3	8.6	8.4	9.9	86.8
1943	3	79.2	9.0	21.3	4.0	47.2	37.9
1944	3	79.3	11.1	19.7	8.6	10.3	68.1
Average		41.0	14.6	7.7	9.5	10.6	50.4

[a] Injury sufficient to lower fruit grade from extra-fancy to fancy or lower by Western grading rules.

[b] Wettable sulphur of 325-mesh except in 1942 when micronized wettable sulphur was used.

for 1942, were commercial brands of approximately 325-mesh fineness used at 8 or 10 pounds per 100 gallons. Commercial spraying equipment was used in all tests. The use of the two fungicides on Anjou pears for 7 seasons in the same orchard gives a fair comparison of their effectiveness in scab control and the degree of grade-reducing russet caused by them (Table 1).

In this series of plots copper phosphate gave about the same control of scab as wettable sulphur, except in 1943, when the control was much greater with copper phosphate. Neither copper phosphate nor wettable sulphur has given satisfactory scab control when the unsprayed check trees have produced approximately 50 per cent or more infected fruits. The distinct advantage of copper phosphate over wettable sulphur under average conditions has been the production of a greater amount of russet-free fruit.

In orchards where scab has been more severe, or when the spray schedule has not been well timed, copper phosphate has controlled scab better than wettable sulphur. The relative merits of the two fungicides in heavily infected orchards are shown in table 2. Observations in the test plots and in commercially sprayed orchards indicated that the fungicidal activity of wettable sulphur sprays was largely dissipated within 10 to 15 days after

TABLE 2.—*Scab control by copper phosphate and wettable sulphur in heavily infected Anjou pear orchards*

Year	Cover sprays	Check trees		Copper phosphate		Wettable sulphur	
		Scab	Russet	Scab	Russet	Scab	Russet
	Number	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*
1933	2	99.4	49.8	72.6	63.0	96.0	81.8
1934	4	82.6	96.7	13.6	94.8	63.1	100.0
1937	3	99.5	.....	38.2	.....	67.8	.....
1940	3	96.9	63.0	26.7	79.1	53.5	68.9
1941	4	96.8	.....	21.3	.....	47.2	.....
Average		95.0	[a]	34.5	[a]	65.5	[a]

[a] Cause of fruit russet obscured by frost damage and other factors.

application, whereas the residue of the copper phosphate mixture was more resistant to weathering and retained its activity longer.

Pears sprayed with copper phosphate have generally produced a smoother skinned fruit than those in any other test plot. The degree of smoothness was much greater than the figures in table 1 indicate. An interesting characteristic of the copper phosphate mixture was its ability to decrease russet injury, even though russeting was obviously due to frost damage, as in 1939 (Table 1). Less fruit russet often occurred in the copper phosphate plots than on unsprayed pears. The reasons for the decreased russet injury in such cases are not understood. On the other hand, in orchards remaining wet for extended periods or during abnormally wet seasons, definite spray russet has resulted from the use of copper phosphate. Results for 1938 and 1944 in table 1, and, as an extreme example, for 1933 in table 2, are illustra-

TABLE 3.—*Effect of wet and dry locations in the production of fruit russet on Anjou pears*

Spray treatment	Cover sprays	Year tested	Total fruit russet	
			Wet orchard	Dry orchard
	Number		*Per cent*	*Per cent*
Copper phosphate	3	1938	47.1	0.04
Wettable sulphur	3	1938	99.4	2.3
Unsprayed trees	0	1939	50.0	7.2
Copper phosphate	2	1939	26.0	0.0
Wettable sulphur	2	1939	51.2	0.4
Unsprayed trees	0	1940	63.0	8.4
Copper phosphate	3	1940	79.1	12.4
Wettable sulphur	3	1940	68.9	14.2

tions of injury caused during wet seasons or in wet orchard locations. The actual percentages for fruit injury in table 1 are not great enough to give a true picture of the injury. In recording the data for 1944, in particular, a good share of the russet injury was on the border line of commercial damage. During most seasons, however, the russet on copper phosphate-sprayed fruit was so slight that the fruit could almost be classed as russet-free. It seems probable that the lime-bentonite mixture is able to neutralize the free copper released from copper phosphate under average weather conditions. With more extreme and extended wet periods, the liberated copper that is constantly present apparently becomes toxic to the fruit skin.

The orchard environment exerts a strong influence on the severity of fruit russet caused by the action of fungicides. Excessive moisture in the form of rain, dews, fog, or very high humidity increases the toxic effects of the fungicides tested. These same factors also favor an increase in scab infections. For convenience of comparison the test plots were classed as wet or dry orchard locations; and the effect of fungicides in causing fruit russet during the same years in similar spray schedules, but in radically different environments, is illustrated in table 3.

The early spray tests showed that copper phosphate gave more effective scab control and less russet on Anjou pears than wettable sulphur. A fungicide more potent against pear scab and less likely to cause injury during wet seasons was still desired for use on spray-sensitive pear varieties.

RECENT SPRAY TESTS

Several of the new organic fungicides offer considerable promise for use on pear trees. For the past 3 years ferric dimethyldithiocarbamate (Fermate) (4) has shown excellent possibilities as a fungicide for pear scab control on the Anjou variety. Results of its use in the same orchard for the 3 seasons are compared with those for other fungicides in table 4.

TABLE 4.—*Control of scab and fruit russet on Anjou pears in the same orchard for 3 years*

Spray treatment[a]	1942		1943		1944	
	Scab	Russet	Scab	Russet	Scab	Russet
	Per cent	*Per cent*	*Per cent*	*Per cent*	*Per cent*	*Per cent*
Check trees—No cover sprays	56.4	5.3	79.2	9.0	79.3	11.1
Copper phosphate 4–4–4–100	8.6	8.4	21.3	4.0	19.7	8.6
Wettable sulphur, 325-mesh, 8–100	.....	.....	47.2	37.9	10.3	68.1
Wettable sulphur, micronized, 8–100	9.9	86.8	36.0	50.2	10.3	71.0
Fermate 1½–100	3.8	2.0	8.0	0.6	5.0	3.6

[a] Four cover sprays applied in 1942; 3 each in 1943 and 1944.

Fermate has given better scab control and caused less injury to Anjou pears than any of the fungicides tested. It has been particularly beneficial during the past 2 seasons when scab infections have been relatively numerous. The Fermate deposit appears to be so persistent and active against the scab fungus that it seems especially desirable to combat early season infections.

The addition of lime to Fermate sprays decreased the value of Fermate for scab control by nearly half during 2 years, and a peculiar ring-spot injury formed in young leaves. While no permanent injury could be detected on tree or fruit, the combination is considered undesirable, especially since lime decreases the persistence of the spray deposit.

Bartlett pear leaves often become more heavily infected by the scab fungus than Anjou leaves, and since these are active infection sources, more fruit infections may develop on Bartlett than on Anjou. On the other hand, Bartlett pear fruit is not very susceptible to spray injury and a russet or other injury not deforming the fruit, does not affect the grade for cannery use. It is therefore possible to use stronger and more effective scab sprays on Bartlett and other spray-tolerant varieties. The comparative value of 3 fungicides in Bartlett pear scab control in a heavily infected orchard during 1944 is shown in table 5. Lime-sulphur was the most effective spray in controlling scab in the heavily infected orchards and probably actually "burned out" many active scab lesions.

TABLE 5.—*Scab control in a heavily infected Bartlett pear orchard during 1944*

Spray treatment (in 3 cover applications)	Fruit scab	Leaf scab	Fruit russet injury
	Per cent	*Per cent*	*Per cent*
Check trees—No cover sprays	94.9	83.0	0.0
Fermate 1½–100	25.5	41.9	0.0
Bordeaux 4–4–100	12.2	11.5	37.2
Lime-sulphur 3–100 in Pink; 2–100 in 2 cover sprays	8.9	7.2	0.0

THE SPRAY SCHEDULE

The number of sprays that must be applied to control pear scab will vary according to weather conditions and the amount of scab present in the orchard. Most fungicides are applied to prevent infections rather than to kill the fungus after it becomes visible on the fruit or leaves. If scab is prevented from developing early in the season, or is held to a small amount, the chances for good commercial control for the remainder of the season are greatly increased. A single spray application may mean the difference between a clean or scabby crop. Micronized wettable sulphur 8 to 100 was applied to pear trees during 1943 and 1944 in a partial schedule. The value of the different spray applications in scab control on Anjou pears during these years is shown in table 6. While 3 applications, pink, calyx, and first cover, gave the best control, they also caused by far the most fruit russet.

Timing is also an important factor in successful scab control. Scab control was poorer during 1943 than in 1944 with the same fungicide, even though unsprayed trees developed almost identical amounts of scab during both years (Table 6). Infections resulted in 1943 when spray coverages were largely dissipated during several rains. During 1944, infection periods occurred only just after sprays had been applied, thereby allowing the spray deposits to exert their maximum influence.

After a good spray material has been properly timed, it is still necessary to apply the fugicide thoroughly. More attention should be directed to applying a large share of the fungicide to the tree tops. It is there that scab control is poorest (1), and a good spray deposit there will give

TABLE 6.—*Scab control and fruit injury resulting on Anjou pears from various applications of wettable sulphur*

Spray applications applied	1943		1944	
	Scab	Russet	Scab	Russet
	Per cent	*Per cent*	*Per cent*	*Per cent*
Check trees—No cover sprays	79.2	9.0	79.3	11.1
Pink spray only	63.8	14.5	47.3	33.4
Calyx spray only	70.0	21.4	59.4	50.2
Pink and calyx spray only	45.2	21.3	26.0	47.6
Pink, calyx, and first cover spray	36.0	50.2	10.3	71.0

considerable protection against infections by redistribution to unprotected parts of a tree during subsequent rains (5). The spray nozzle pressure should be cut down on high-pressure equipment so that only a "fog" is applied to the lower parts of the tree. This is particularly important when dirty water or coarse particles of spray material are in the spray tank. Specific data are lacking, but both writers have frequently observed that considerable russet may be caused on tender-skinned pears by driving fine particles of silt or coarse spray particles against the fruit surface under high pressure. The relation between scab control and injury caused by wettable sulphur (8 to 100) on Anjou pears at various heights in the tree is shown in table 7.

TABLE 7.—*Relation of the height of fruit on the tree to scab control and russet injury on Anjou pears following wettable sulphur spray*

Fruit location[a]	Scab	Russet
	Per cent	*Per cent*
Bottom third of tree	10.0	62.0
Middle third of tree	14.5	27.3
Top third of tree	23.2	16.9

[a] Trees averaged approximately 25 feet in height.

EFFECT OF FUNGICIDES ON FOLIAGE COLOR AND FRUIT SET

It was noticed during the early spray tests that sulphur fungicides usually caused Anjou pear leaves to remain yellower throughout the season than leaves on trees sprayed with other materials. A number of growers also suggested that the use of sulphur was decreasing their yield of fruit. Tests were started in 1942 to evaluate these observations. The data are not complete enough to make final statements, but certain trends requiring closer study have been indicated.

Trees sprayed with copper phosphate or with Fermate and those left unsprayed developed distinctly greener foliage than sulphur-sprayed trees. These reactions have been consistent during 3 years, although the degree of color difference has varied somewhat with the seasons. The finer sulphurs appear to be more toxic to the trees than the coarser forms, but the orchard environment, rootstocks, and other factors probably influence the extent of injury. Leaf color has been improved most in the copper phosphate plots. Kadow and Anderson (6) reported a similar reaction in cherry trees after the use of copper phosphate.

Large differences in the average fruit yield occurred on the test trees in 1942. Trees sprayed with micronized wettable sulphur produced only about 1/3 as much fruit as the check trees or those sprayed with copper phosphate or with Fermate. The total blossoms were counted on test trees during 1943 and 1944 and the actual fruit set was determined from the total fruits harvested. The crop in 1943 was too light to provide reliable information. In 1944, the set of fruit varied too greatly among trees with

the same treatment to permit definite conclusions, but again the micronized sulphur seemed to reduce the set of fruit below that of the other plots.

SUMMARY AND CONCLUSIONS

A spray material that is more effective against pear scab and less toxic to Anjou and other spray-sensitive pears has been needed to replace the wettable sulphurs now used because of the lack of better fungicides. A fungicide is often needed for scab control during the summer, but sulphur materials have 4 evident disadvantages for use on Anjou pears during that period. 1. They may cause a severe sun-scald type of injury on fruits when temperatures rise above 90° F. 2. Insecticides containing oil may not be applied within 45 days after sulphur fungicides on pear trees for fear of causing severe leaf-spotting or defoliation. This fact seriously hampers the control of late-season spider mite attacks in scabby orchards. 3. Sulphur is dissipated rapidly during warm weather and is effective in scab control for only relatively short periods. 4. Sulphur apparently is directly toxic to Anjou pear trees.

Lime-sulphur was the most satisfactory fungicide tested for pear scab control on those varieties not subject to spray injury.

Copper phosphate has given equal or better scab control than wettable sulphur in most cases, and caused much less fruit russet during average weather conditions. During wet seasons it has not given adequate scab control and it produced excessive fruit russet. The appearance of foliage sprayed with copper phosphate has been superior to that in any other spray plot. This material has not reduced fruit set and has been compatible with most insecticides. It has not been available during the war, and the mixture is so bulky that growers object to it.

Fermate seems to be an immediate substitute for the wettable sulphurs for use on spray-sensitive pear varieties. It has given consistently good scab control and has not injured fruit for the past 3 years, and foliage color has been excellent on sprayed trees. Fermate has given no trouble in most mixtures and oil may be safely used in the same application or soon afterwards.

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THE RELATION OF THE OCCURRENCE OF FOLIAGE SYMPTOMS OF CHLOROTIC STREAK OF SUGAR CANE TO THE DISTRIBUTION OF THE VIRUS IN THE PLANT

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(Accepted for publication June 4, 1945)

INTRODUCTION

Foliage symptoms of chlorotic streak of sugar cane are inconstant. Although some infected plants may manifest them throughout their period of growth, frequently the characteristic leaf streaks are present only intermittently. There is little information in the literature as to the relation of this inconstancy of foliage symptoms to the occurrence of the virus in the stalk or to actual recovery from the disease. Whether, for example, the foliage "recovery" resulting from the senescence of old, streaked leaves, and the absence of streaks on the new foliage subsequently produced, is related to disappearance of the virus from the stalk, has not been previously investigated. A knowledge of these points is of practical value in studying control measures, as well as from the standpoint of gaining a better understanding of the disease itself, and it was to obtain information regarding them that these investigations were undertaken.

Thus far, important losses from chlorotic streak in Louisiana have been limited to relatively small areas where the virus became established before control measures were undertaken. Nevertheless, it is apparent from the losses already incurred, and from experimental results showing the severe reductions in germination of seed cuttings and ratoons that may occur (2, 3, 4), that the disease is of considerable potential importance. The fact that it can be held in check in moderately susceptible varieties by roguing seed plots (1, 2, 3), or eliminated from seed-cane cuttings by hot-water treatment (5, 8, 9, 11), lessens somewhat its threat to sugar-cane culture in areas where it has become established.

LOSS OF FOLIAGE SYMPTOMS

The loss of foliage symptoms from chlorotic-streak-infected sugar-cane plants has been noted previously (3, 10, 11). In the present study, this resulted principally from the death by senescence of the leaves on which the streaks occurred. Foliage symptoms were lost sometimes when normal green color was restored to indefinite, faintly chlorotic areas on infected leaves, but well-defined streaks did not disappear from living leaves.

During 1940 and 1941, loss of foliage symptoms in 4 varieties of sugar cane, the C.P. numbers 807, 28/19, 29/103, and 29/320, and its relation to the occurrence of the virus in the lateral buds of affected stalks, was studied

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in the field. Young plants obtained from diseased seed cuttings of each variety, and showing definite leaf streaks, were marked with numbered stakes in June. At intervals of approximately 15 days during the remainder of the growing season, leaf symptoms on the primary and secondary shoots were recorded. Each shoot in the stool was labeled with a numbered metal tag, the primary shoot being designated as number 1, and the tillers, or secondary shoots, numbered consecutively as they arose. In 1940, 100 stools each of C.P. 28/19 and C.P. 29/320 were studied, and in 1941, 100 of C.P. 29/320, 25 of C.P. 807, and 16 of C.P. 29/103.

The percentage of individual shoots with either temporary or permanent loss of foliage symptoms at some time during the season, and of secondary shoots remaining permanently free of such symptoms, are shown in table 1.

TABLE 1.—*Percentage of primary and secondary shoots of 4 varieties of sugar cane showing temporary or permanent loss, or absence of foliage symptoms of chlorotic streak*

C.P. variety	Loss of symptoms										Secondary shoots permanently without symptoms
	Temporary					Permanent					
	Shoots										
	Primary	Secondary				Primary	Secondary				
		1	2	3	4		1	2	3	4	
807	20.0	.....	6.0	.....	.....	32.0	18.0	12.5	33.3	.....	44.4
28/19	16.5	12.3	1.8	.....	.....	8.8	13.7	5.4	2.9	.....	11.7
29/103	6.7	.....	20.0	7.7	.....	6.7	.....	7.7	.....	.....	8.7
29/320[a]	4.3	2.2	1.2	.....	1.6	7.5	.....	5.9	2.7	7.9	8.5
29/320[b]	16.8	3.3	.....	.....	.....	6.3	1.0	2.3	.....	.....	7.7

[a] 1940.
[b] 1941.

Some plants of each variety had symptoms initially but became symptomless at some time during their growth. For the most part this temporary loss of symptoms occurred during the period of rapid growth in July and August, when the initially streaked leaves were lost through senescence and the new foliage produced was symptomless. During this time the new foliage also lacked the abnormal straightness and stiffness that characterizes many young plants in the field earlier in the season.

There was a tendency for primary shoots to lose symptoms more often than secondary shoots, although there were exceptions; and in C.P. 29/103 more of the secondary shoots recovered. Generally, loss of symptoms in primary shoots was by senescence of the affected leaves, whereas secondary shoots did not reach senescence during the period of observation.

Secondary shoots arising in diseased stools of C.P. 807 are much less likely to be invaded than are those of the other 3 varieties. All of the symptomless secondary shoots of C.P. 807 produced virus-free plants when indexed in the greenhouse, whereas diseased plants were produced from some of the symptomless secondary shoots of the other varieties. Recovery from

foliage symptoms in infected shoots was more often permanent in C.P. 807 than in the other varieties.

DISTRIBUTION OF CHLOROTIC STREAK IN THE LATERAL BUDS OF INFECTED STALKS

According to Abbott (1), Wilbrink (11), and Bell (6, 7), not all of the lateral buds on chlorotic-streak-infected stalks of sugar cane give rise to diseased shoots when planted. However, the author's previous observations in this respect, and presumably those of Wilbrink and Bell, were on plants growing in the field where secondary spread of the virus was probably occurring, thus making impossible an exact determination of the extent of so-called recovery from the virus, and the distribution of infected buds on the stalk. The object of the present study was to obtain this information in an insect-proof greenhouse, and under conditions eliminating the possibility of secondary spread. Four commercial varieties of sugar cane grown in Louisiana, C.P. 807, 28/19, 29/103 and 29/320, were used, all of which have been observed to produce apparently healthy plants from diseased seed cuttings in the field.

Experiment 12. Two varieties, C.P. 28/19 and 29/320, were included in experiment 12, which was conducted during 1940–41. The final readings were made in late October and the symptoms estimated as mild if the leaf streaks were few or small and as severe if they were numerous, large, or necrotic. The stalks that had produced at least 5 mature lateral buds were harvested and taken to the greenhouse for indexing. There they were cut into single-bud cuttings, and the buds on each stalk numbered consecutively from the base upward. Missing and damaged buds were included in the seriation. The cuttings were potted in steamed, sandy-loam soil in 4-inch clay pots that had been immersed for 2 hours in a 1–250 solution of formaldehyde and placed on greenhouse benches where the air temperature was maintained at a minimum of 75° F.

Experiment 23. In experiment 23, which was conducted during 1941–42, 100 stools of C.P. 29/320, 25 of C.P. 807, and 16 of C.P. 29/103 were marked and observed as described under experiment 12. On 75 plants of C.P. 29/320, a record was kept of the total number of living leaves and the number with streaks at each observation date, with the view of obtaining a more accurate measure of symptom severity than the estimate made in experiment 12. In the fall the stalks were harvested and indexed. The buds of C.P. 29/320 were potted on September 22, 1941, and the plants not showing the disease were discarded on March 10, 1942. Those of C.P. 807 and 29/103 were potted on October 29 and 30, 1941, and discarded on July 3, 1942. All had been ratooned once.

It is possible that in these experiments additional plants would have eventually developed symptoms of chlorotic streak if they could have been held for longer periods, although it is improbable that the number would have been such as to affect the results materially. This was indicated by the

fact that 10 plants of C.P. 29/320 from each experiment that were retained until they had produced jointed stalks, remained symptomless, as did the shoots produced when the lateral buds from these stalks were planted.

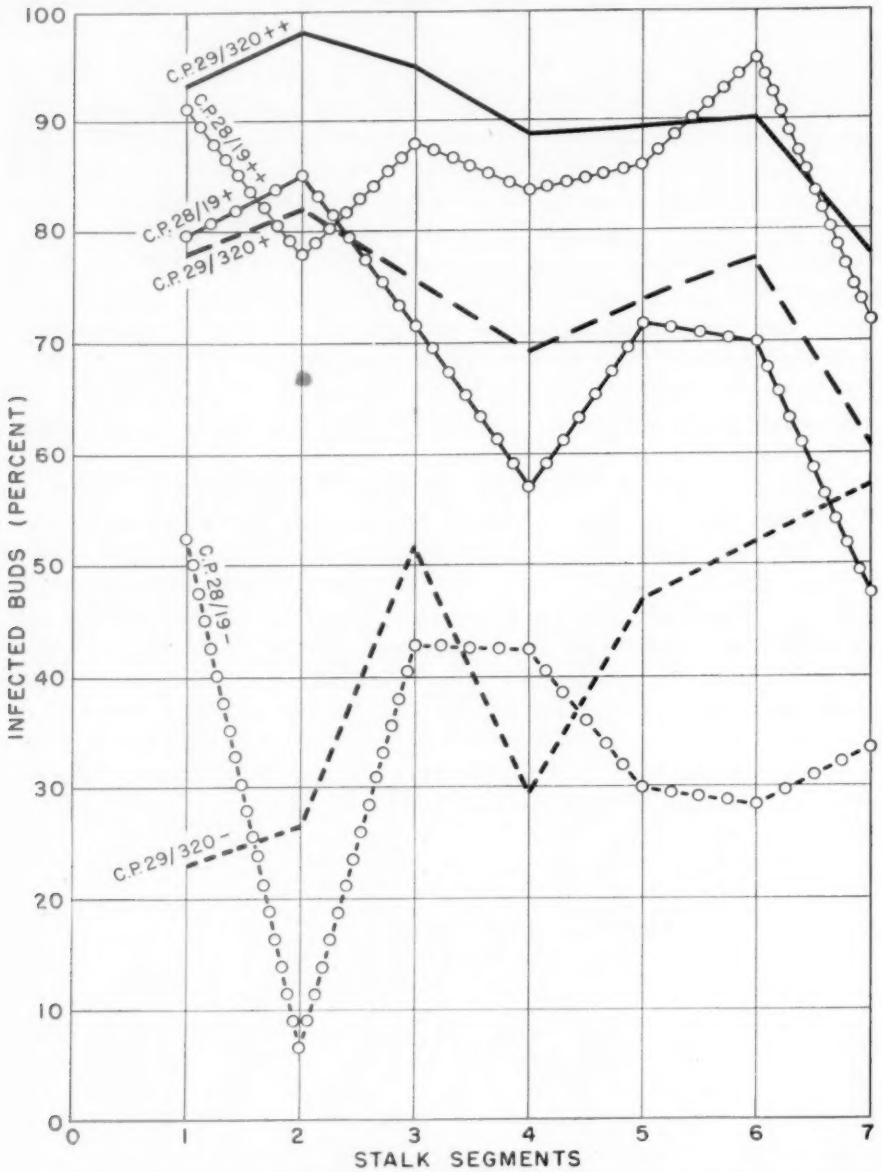

FIG. 1. Percentage of infected buds in segments of stalks of C.P. 28/19 and 29/320, classified according to severity of symptoms (Experiment 12): −, symptomless plants; +, mild symptoms; ++, severe symptoms.

For analysis of the results with respect to distribution of the virus in the lateral buds, each stalk was divided into 7 segments, each of which consti-

tuted approximately 14.3 per cent of the entire stalk. The object of such a division on a percentage basis was to place buds on different stalks of pre-

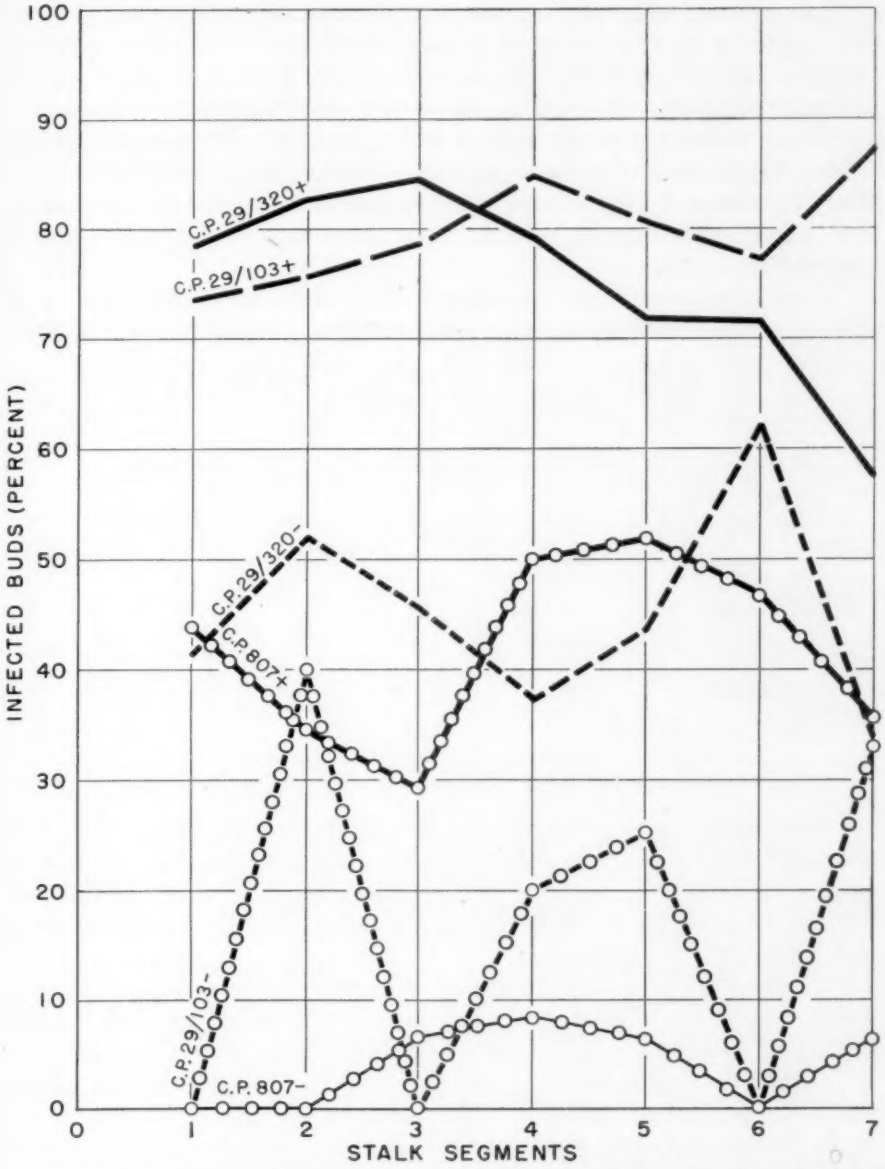

FIG. 2. Percentage of infected buds in segments of stalks of C.P. 807, 29/103, and 29/320, classified according to presence or absence of symptoms (Experiment 23): –, symptomless plants; +, plants with symptoms.

sumably similar physiological and anatomical development in the same relative groups. For example, bud number 10 on a long stalk of 15 to 18 buds might differ physiologically from bud number 10 on a shorter stalk on which

it was the top bud. The results of the experiments are presented graphically in figures 1 and 2.

In experiment 12 (Fig. 1) there was a tendency for greater freedom from the virus in the upper portions of the stalks that had leaf symptoms. Otherwise there was no consistent tendency for the virus to be localized in any portion of the stalk. In both experiments, distribution in the symptomless plants was more erratic than in those with symptoms. There was a definite relationship between severity of symptoms and the number of infected buds. The symptomless stalks produced the lowest number of diseased plants, those with severe symptoms the highest, while those with mild symptoms were intermediate.

These relationships are emphasized by the frequency distribution in table 2, in which the data are arranged according to the percentage of buds

TABLE 2.—*Distribution of stalks of 2 varieties of sugar cane in frequency classes of percentages of buds per stalk producing diseased plants. Experiment 12, stalks indexed in the greenhouse*

Variety and symptom severity in field when harvested	Distribution of stalks in frequency classes[a]						
	0	1–20	21–40	41–60	61–80	81–99	100
	Pct.	*Pct.*	*Pct.*	*Pct.*	*Pct.*	*Pct.*	*Pct.*
C.P. 28/19							
Symptomless	41	6	6	12	18	12	6
Mild	.....	8	8	15	35	19	15
Severe	.....	.....	.....	9	16	28	47
C.P. 39/320							
Symptomless	19	6	31	6	25	6	6
Mild	2	.....	9	9	18	39	23
Severe	.....	.....	.....	3	5	49	44

[a] Classes based on percentage of buds per stalk producing diseased plants.

per stalk producing diseased plants. With both varieties, nearly half of all the stalks in the group with severe symptoms produced diseased plants from all of their buds. In the symptomless group, 41 per cent of the stalks of C.P. 28/19 produced all healthy plants, whereas only 19 per cent of those of C.P. 29/320 were healthy.

That severity of foliage symptoms at harvest time was related to virus invasion of the buds was further indicated by the record made of the percentage of streaked leaves on 75 plants throughout the season in experiment 23. When the results obtained were arranged in frequency classes according to the percentage of leaves with streaks at the time of harvest, it was found that on stalks with 1 to 20 per cent of their leaves streaked, 61 per cent of the buds produced diseased shoots; in the groups with 21 to 40 per cent, and 41 to 60 per cent of the leaves streaked, 81 per cent of the buds in each were diseased; and in the group with 61 to 80 per cent of the leaves streaked, 95 per cent of the buds were diseased.

Nevertheless, the occurrence of streaks on a leaf did not necessarily indicate infection of the bud on the node subtending it. Some stalks with dis-

seased leaves produced only apparently healthy plants when their lateral buds were germinated. Symptoms on individual leaves of 6 plants of C.P. 29/320 in the greenhouse were recorded from germination until plants were about 6 months old and had produced 7–10 lateral buds of sufficient maturity to germinate. From buds subtending diseased leaves 19 diseased and 13 healthy plants were produced; and from buds subtending healthy leaves 2 diseased and 4 healthy plants were produced.

There was no consistent relationship between the percentage of germination of buds on stalks in the different symptom groups and the occurrence of chlorotic streak. Because of the known adverse effect of the virus on germination in the field, it might have been expected that stalks or segments with the lowest incidence of infection would have the highest germination. This, however, was not true, the average germination being approximately the same regardless of symptoms. This may have been at least partially the

TABLE 3.—*Recovery from chlorotic streak in foliage and buds of entire stools of 4 varieties of sugar cane*

C.P. variety	Expt. No.	Total No. of stools	Stools showing complete recovery in:		
			Foliage		Buds
			Temporary[a]	Permanent	
			Pct.	*Pct.*	*Pct.*
807	23	25	33.3	25.0	16.7
28/19	12	100	17.0	5.0	2.0
29/103	23	16	0.0	0.0	0.0
29/320	12	100	7.0	5.0	2.0
29/320	23	100	6.0	1.0	4.0

[a] Foliage of all shoots of the stool symptomless at some time, developing symptoms again later in the season.

result of the favorable temperature for rapid germination of sugar cane in the greenhouse, at which the virus had no marked influence on germination.

Recovery in the Buds. The results were examined for evidence of recovery from the disease in the buds, aside from the loss of foliage symptoms, which was discussed earlier. Some of the data obtained may be interpreted as evidence of such recovery. In table 3, data from experiments 12 and 23 are presented on the basis of entire stools, rather than for individual shoots (as in table 1); and in table 4, the records of symptoms in the field and the number of lateral buds producing diseased plants in the greenhouse are summarized for 9 typical stools.

Some of the stools of C.P. 807, 28/19, and 29/320 showed complete loss of foliage symptoms in all of their shoots, and in some of these the virus was apparently not present in the lateral buds at harvest time, as evidenced by the absence of symptoms in the plants grown from them (Table 4). Examples are C.P. 807, stool 7, C.P. 28/19, stool 20, and C.P. 29/320, stool 56 (Table 4). This is evidence of recovery if it can be assumed that the buds actually had been invaded by the virus. That such an assumption is justi-

fied is indicated by the fact that in experiments 12 and 23, 83 per cent of the stalks of C.P. 807 with foliage symptoms at some time during the season were found to have some infected buds, 93 per cent of those of C.P. 29/103, 95 per cent of those of C.P. 29/320, and 99 per cent of those of C. P. 28/19.

TABLE 4.—*Occurrence of chlorotic streak in leaves and buds of individual stalks of 3 varieties of sugar cane*

C.P. variety[a]	Stool and shoot No.[b]	Leaf symptoms[c]						Buds indexed	
		May	June	July	Aug.	Sept.	Oct.	Total	No. with C.S.
807	7–1	.....	+	+	+	–	–	12	0
	7–2	.....	.....	–	–	–	–	12	0
	7–3	.....	.....	.....	–	–	–	10	0
	11–1	.....	+	+	–	–	+	16	0
	11–2	.....	–	–	+	–	–	10	0
	11–3	.....	–	+	+	+	+	4	4
	11–4	.....	.....	–	+	–	–	8	0
	17–1	.....	+	+	+	+	+	15	6
	17–2	.....	.....	.....	+	–	–	6	0
28/19	20–1	.....	–	–	–	–	.....	11	0
	20–2	.....	+	–	–	–	.....	9	0
	20–3	.....	+	+	+	–	.....	9	0
29/320	6–1	+	+	–	–	–	.....	6	6
	6–2	+	+	+	+	+	.....	4	4
	56–1	+	+	+	–	–	.....	12	0
	56–2	.....	–	–	–	–	.....	8	0
	56–3	.....	.....	–	–	–	.....	5	0
	57–1	+	+	+	+	+	.....	13	11
	57–2	.....	–	–	–	–	.....	11	11
	57–3	.....	–	–	–	–	.....	8	8
	57–4	.....	–	–	–	–	.....	4	3
	82–1	+	+	–	+	+	.....	9	3
	82–2	.....	–	–	–	–	.....	6	0
	82–3	.....	–	–	–	–	.....	8	0
	82–4	.....	.....	.....	–	–	.....	9	0
	96–1	+	+	+	–	–	.....	9	0
	96–2	.....	–	–	+	+	.....	7	0
	96–3	.....	.....	–	+	+	.....	6	4
	96–4	.....	.....	–	–	+	.....	4	0
	99–1	+	+	+	+	–	.....	8	0
	99–2	.....	–	–	–	–	.....	9	2
	99–3	.....	–	–	+	+	.....	8	6
	99–4	.....	.....	–	–	+	.....	4	3

[a] Material of C.P. 28/19 was from experiment 12; all other material was from experiment 23.

[b] The primary shoot in each stool is no. 1; the secondary shoots, 2 to 4, according to the order in which they arose in the stool.

[c] + indicates symptoms present; – indicates symptoms absent.

That the loss of foliage symptoms was associated with a decrease in incidence of the virus in the buds is further shown by table 5, in which data from experiments 12 and 23 are presented on the basis of the occurrence of chlorotic streak in the buds in relation to the constancy of leaf symptoms during the period from May to October. On the whole, the buds of plants showing leaf symptoms constantly throughout the season were invaded to a greater extent than those showing foliage recovery at some time. There were excep-

tions to the general rule, however, for some individual plants that had leaf symptoms at only the final reading produced diseased plants from all of their lateral buds, and others that were symptomless for as long as 3 months produced infected plants from a majority of their buds (Table 5). The groups of stalks of C.P. 29/320 that were symptomless constantly in experiments 12 and 23 (all secondary shoots) also showed a higher percentage of diseased buds than did those that had symptoms at some time, but had recovered by harvest. While the number of stalks studied in these groups was too small for the results to be considered conclusive, these data, together with those for the symptomless stalks, indicate that stalks of C.P. 29/320 are more likely to be infected with the chlorotic-streak virus without showing leaf symptoms, than are those of the other 3 varieties studied.

TABLE 5.—*Occurrence of chlorotic streak in lateral buds of 4 varieties of sugar cane in relation to the constancy of leaf symptoms*[a]

C.P. variety	Expt. No.	Occurrence of leaf symptoms during the season[b]							
		+ continuously		Alternating + and − but + at harvest		Alternating + and − but − at harvest		− continuously	
		Stalks	Buds C.S.	Stalks	Buds C.S.	Stalks	Buds C.S.	Stalks	Buds C.S.
		No.	*Pct.*	*No.*	*Pct.*	*No.*	*Pct.*	*No.*	*Pct.*
807	23	21	49.8	5	34.8	17	15.2	11	3.9
28/19	12	90	77.1	6	45.5	12	49.5	6	7.8
29/103	23	39	83.1	4	55.1	2	40.0	2	0
29/320	12	79	81.5	4	78.0	12	33.6	4	70.6
29/320	23	70	77.0	15	63.4	9	41.9	13	45.0

[a] Includes only those stalks observed for at least 3 months.
[b] + indicates symptoms present; − indicates symptoms absent.

It is possible that in the recovered plants the virus had been present only in leaf and stalk tissues, and had not invaded the lateral buds. It will be noted in table 4, for example, that several stalks with leaf symptoms in the field produced apparently healthy plants when their buds were planted in the greenhouse. Later, when buds on stalks grown from these plants were germinated, only apparently healthy plants were produced. It is possible that the buds on the original plants were never invaded by the virus.

In the absence of some means of definitely determining the presence of the virus in the plant other than the occurrence of the leaf streaks, it is difficult to obtain positive proof of recovery. Considering all of the evidence, however, it seems reasonable to assume that at least some of the lateral buds on the stalks that apparently recovered had been invaded by the virus, and that the absence of the disease in the plants produced from them is indicative of recovery during or preceding germination.

That the stalk and not the stool is the physiological unit involved in the distribution of the virus in the plant, or in recovery from the disease, is evi-

dent from the data in table 4. The disease appeared in or disappeared from individual stalks of a stool irrespective of other stalks comprising the stool.

MOVEMENT OF THE VIRUS WITHIN THE STOOL

Natural Movement. The field studies indicated that chlorotic-streak virus spreads from infected to healthy shoots comprising a stool of sugar cane (Table 4). However, since the field-grown plants were exposed to secondary spread by insects, it was uncertain whether the appearance of the disease in previously healthy shoots of a diseased stool resulted from transfer by insects, or from movement into that shoot from another diseased shoot. Proof of such movement has been obtained with plants grown from diseased single-bud cuttings in steamed soil in an insect-proof greenhouse. The plants were started in 4-inch pots and later transferred to 4-gallon cans, where they remained until several tillers had produced jointed stalks with mature buds. The results with several plants of the variety C.P. 29/320 are presented in table 6.

TABLE 6.—*Occurrence of chlorotic streak leaf symptoms in secondary shoots arising from diseased primary shoots of the sugar-cane variety C.P. 29/320*

Plant No.	Symptoms[a] in				
	Primary shoot	Secondary shoots			
		1	2	3	4
12–2–1	+	+	−	.....	.....
12–2–9	+	+	+	.....	.....
40–1–1	+	+	+	−	−
40–1–10	+	−	+	.....	.....
40–3–10	+	−	−	−	.....
85–3–6	+	+	+	+	.....
II–D–93	+	+	−	+	.....

[a] + indicates symptoms present; − indicates symptoms absent.

The virus may spread to all of the secondary shoots (Plant No. 85–3–6), to none of them (Plant No. 40–3–10), or to some and not to others. That the symptomless secondary shoots were not invaded was indicated when the mature lateral buds produced on them were planted in the greenhouse, and the resulting plants remained free of chlorotic streak symptoms. It is possible that the apparently symptomless shoots were invaded, but that the concentration of virus was insufficient to induce the characteristic leaf symptoms.

Effect of Severing the Primary Shoot. Removing the primary shoot from a stool of sugar cane usually stimulates the development of new secondary shoots and the growth of those already present. Presumably food materials stored in the underground portion of the stem of the primary shoot move into the secondary shoots in this process. The percentage of plants with chlorotic-streak symptoms is generally greater in fields of ratoons than in the same fields when they were in plant cane, and it is possible that the appear-

ance of the disease in the shoots arising following ratooning may be correlated with movement of reserve food materials into them.

To determine the effect of severing the infected primary shoot on the development of chlorotic streak in secondary shoots previously showing no indication of the disease, 6-month-old plants of the variety C.P. 29/320 growing in 4-gallon cans in the greenhouse were selected, all of which showed chlorotic streak symptoms in the primary shoot, but none in any of the secondary shoots. There were from 1 to 6 secondary shoots per plant and each shoot had from 4 to 10 unfolded leaves. The primary shoot was cut from some of the plants immediately above the point of origin of the first secondary shoot, and the others were left as controls. At intervals thereafter notes were made on the occurrence of chlorotic streak in the secondary shoots. The results of 2 typical experiments are presented in table 7.

TABLE 7.—*Effect of severing the primary shoot on the appearance of chlorotic streak symptoms in the secondary shoots of stools of the variety C.P. 29/320*

Duration of expt.	Treatment	Number of stools	Secondary shoots				
			At beginning of expt.		At end of expt.		
			Total No.	With C.S.	Total No.	With C.S.	
Days				No.		No.	Pct.
90	Control	10	16	0	54	6	11.1
	1 shoot severed	10	28	0	57	32	56.1
30	Control	6	15	0	15	6	40.0
	1 shoot severed	8	18	0	21	16	76.2

It is apparent that removing the infected primary shoot stimulated the movement of the virus into the secondary shoots. Approximately twice as many in one experiment, and 5 times as many in the other, developed symptoms of the disease in the treated as in the untreated plants. Similar results were obtained when the primary shoot was removed from plants grown from infected seed cuttings, but in which all shoots had remained symptomless up to the time the primary shoot was severed.

Since reserve food materials presumably moved from the underground portion of the stem into the secondary shoots following the cutting of the primary shoot, it may be assumed that the appearance of chlorotic streak in the secondary shoots was correlated with this movement. Such an assumption offers a plausible explanation for the commonly observed higher incidence of chlorotic streak in ratoons as compared with plant cane in the field. Presumably this results from the development of symptoms in plants that are infected as plant cane, but in which foliage symptoms do not appear until they are ratooned. It is recognized that the plants in these experiments, in which infection was primary, *i.e.*, from the seed cutting, were not strictly comparable with field-grown plants which become infected through secondary spread by insect vectors. However, in both instances, the physiological

process of ratooning and the consequent movement of the virus into the new shoots would be analogous.

DISCUSSION

The studies reported in this paper explain the often-observed transient nature of chlorotic streak in the field, showing that the apparent decline in incidence of the disease during the season of rapid sugar-cane growth in summer results from loss of foliage symptoms through the senescence of streaked leaves and the absence of streaks in the new foliage. In general, this was associated with a reduction in the number of infected buds, but complete recovery in both foliage and buds apparently occurred in a relatively small percentage of the stools studied. The majority of the plants were only transiently symptomless.

From a practical standpoint, the significance of the results lies in showing the desirability of roguing seed-cane plots for chlorotic streak early in the season, *i.e.*, in May and June, before the rapid summer growth begins, and before many infected plants lose external symptoms of the disease. Although the percentage of completely symptomless infected stools at any one time during the season was not high for C.P. 29/320, it was considerable for C.P. 28/19 and C.P. 807 (Table 3). Considering also the greater difficulty of detecting the often inconspicuous symptoms in the larger cane during the summer, as well as the greater expense resulting from the increased time required for inspecting the taller cane, the importance of early roguing is further apparent. Practical experience has shown that if the diseased stools are eliminated in May and June, infection may be reduced to a very low point without summer roguing (1, 2, 3).

The sugar-cane varieties studied differed in the extent to which shoots and buds of infected plants were diseased. This may have been the result of more restricted movement of the virus in the plants of some varieties, a greater degree of recovery, or both. These differences in relative degree of distribution of the virus were not correlated with relative susceptibility to infection. C.P. 807, for example, which is about as susceptible to infection as C.P. 28/19 (4), recovered from foliage symptoms to a considerably greater degree than the latter variety, and the virus was present in a lower percentage of the secondary shoots and lateral buds. Likewise, C.P. 29/320, which is near C.P. 29/103 in susceptibility to infection (4), showed a greater degree of foliage recovery and a lower percentage of infected lateral buds. Varieties of equal susceptibility to infection also differ in the extent to which their germination and yields are reduced by the disease (4). It is apparent, therefore, that a true appraisal of the importance of the disease in a variety must take into account its ability to recover from and to resist the deleterious effects of the virus, as well as its susceptibility to infection.

The possibility that sugar-cane plants may harbor the virus of chlorotic streak without showing the characteristic leaf streaks, introduces an element of uncertainty into experimental work, the results of which depend on deter-

mining the presence or absence of the virus in a plant. Admittedly, some of the results reported in this paper may have been influenced to some extent by this uncertainty. It was found, however, that in the greenhouse, leaf symptoms could be induced in many plants that were previously symptomless, by a heavy application of $NaNO_3$. Plants that failed to develop symptoms following this treatment did not subsequently show any indication of the presence of chlorotic streak, even though some of them or their progeny were carried for 2 years in the greenhouse. It is believed, therefore, that the number of plants in these experiments that may have harbored the virus without showing symptoms is very small, and that the results were not affected materially thereby.

SUMMARY

Chlorotic-streak-infected sugar-cane plants became symptomless through the loss by senescence of the streaked leaves, and the temporary or permanent failure of symptoms to develop in the foliage subsequently produced. Less frequently, foliage recovery occurred through the restoration of the normal green color in faintly chlorotic areas of diseased leaves, but disappearance of well-defined streaks from affected tissues was not observed.

On the whole, the extent of infection of the buds was correlated with the severity of foliage symptoms, and was greater in those plants that had symptoms continuously than in those that showed temporary or permanent loss of symptoms. However, loss of foliage symptoms was not necessarily associated with disappearance of the virus from the stalks: in some plants the presence of the virus in the buds was demonstrated several months after the leaf symptoms disappeared.

Some buds from infected stalks produced apparently healthy plants on germination. It is assumed that this was the result of actual recovery from the disease, although it is possible that these buds had not been invaded by the virus, or that the concentration of virus was insufficient to induce the characteristic leaf symptoms. There was a tendency for the buds on the upper segments of the stalks to have greater freedom from the disease than the lower.

The sugar-cane varieties studied differed in the extent to which shoots and buds of infected plants were diseased. This may have been the result of a more restricted movement of the virus in the plants of some varieties, a greater degree of recovery, or both.

The stalk rather than the stool of sugar cane was found to be the physiological unit involved in the distribution and movement of the virus in the plant. The disease appeared in or disappeared from individual stalks independently of the rest of the stool.

Removing the infected primary shoot from a stool of cane with initially healthy secondary shoots caused a marked increase in the number of diseased secondary shoots. It is postulated that the appearance of the disease in the secondary shoots may have been correlated with the movement of reserve materials into them from the underground portion of the stem of the infected

primary shoot. This offers a plausible explanation for the greater incidence of the disease in ratoons as compared with plant cane.

DIVISION OF SUGAR PLANT INVESTIGATIONS,
U. S. DEPARTMENT OF AGRICULTURE.

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PHYTOPATHOLOGICAL NOTES

Virus Diseases of Guayule.—The reactions of guayule seedlings to a number of plant viruses have been tested experimentally.

Ordinary tobacco-mosaic virus. Guayule seedlings were experimentally infected with ordinary tobacco-mosaic and tobacco-ring-spot viruses by mechanical inoculation using the carborundum method. Seedlings inoculated with the tobacco-mosaic virus developed small necrotic areas on the inoculated leaves but not on those newly developing. The virus was recovered and transferred to *Nicotiana glutinosa* from the inoculated leaves but not from the newly developing leaves. Local lesions appeared on the inoculated leaves of *N. glutinosa.* The infection of guayule seedlings was local and not systemic.

Tobacco-ring-spot virus. Several hundred guayule seedlings inoculated with the tobacco-ring-spot virus were symptomless carriers of the disease. The virus was recovered from the inoculated leaves and transferred to *Nicotiana glutinosa.* The infection was local and not systemic.

Cucumber-mosaic viruses. Fifty-five potted guayule seedlings were nonsusceptible to western cucumber-mosaic virus, and 30 seedlings were nonsusceptible to the ordinary cucumber-mosaic virus. In flats containing approximately 100 plants, no infection was observed.

Celery-mosaic viruses. There was no infection in 50 potted guayule seedlings inoculated with virus of western-celery-mosaic, nor in 25 seedlings inoculated with celery-calico virus. Plants growing in flats also were nonsusceptible.

Beet-mosaic virus. Twenty-five potted guayule seedlings were non-susceptible to the beet-mosaic virus, as were approximately 100 plants grown in flats and inoculated with this virus.

Sugar-beet curly-top virus. Lots of 20 infective beet leafhoppers, *Eutettix tenellus* (Baker), were used to inoculate guayule seedlings with the curly-top virus but no symptoms of the disease developed. Noninfective beet leafhoppers failed to recover the virus and transfer it to healthy sugar beets. Nymphs which hatched from eggs deposited in guayule seedlings died.

California-aster-yellows virus. Attempts were made to infect guayule seedlings with the California-aster-yellows virus. The following species of leafhoppers, vectors of this virus, were used to inoculate the plants and noninfective adults of each species failed to recover the virus and transfer it to healthy aster and celery plants: short-winged aster leafhopper, *Macrosteles divisus* (Uhl.); long-winged aster leafhopper, a race of the same species, *M. divisus;* mountain leafhopper, *Colladonus montanus* (V. D.); geminate leafhopper, *Idiodonus geminatus* (V. D.); *Phlepsius lathropi* Baker, *P. latipex* De L., *P. spatulatus* V. D., *Acinopterus angulatus* Law., and *Cloanthanus irroratus* (V. D.).

Toxic salivary secretion. The toxic salivary secretion of *Xerophloea vanduzeei* De L. induces some of the symptoms closely resembling curly top on sugar beets and aster yellows but this leafhopper failed to produce symptoms on guayule seedlings.—HENRY H. P. SEVERIN, Guayule Research Project, Bureau of Plant Industry, United States Department of Agriculture and College of Agriculture, University of California.

The Sooty Stripe Disease of Sorghum.—The fungus, *Titaeospora andropogonis* (Miura) Tai, described[1] as the cause of a serious leaf disease of Kaoliang sorghum in China and Manchuria, was first reported in the United States in 1942.[2] Since then the fungus has apparently been spreading in Louisiana and Mississippi and at this time it seems advisable to present the information which is now available concerning the disease it causes.

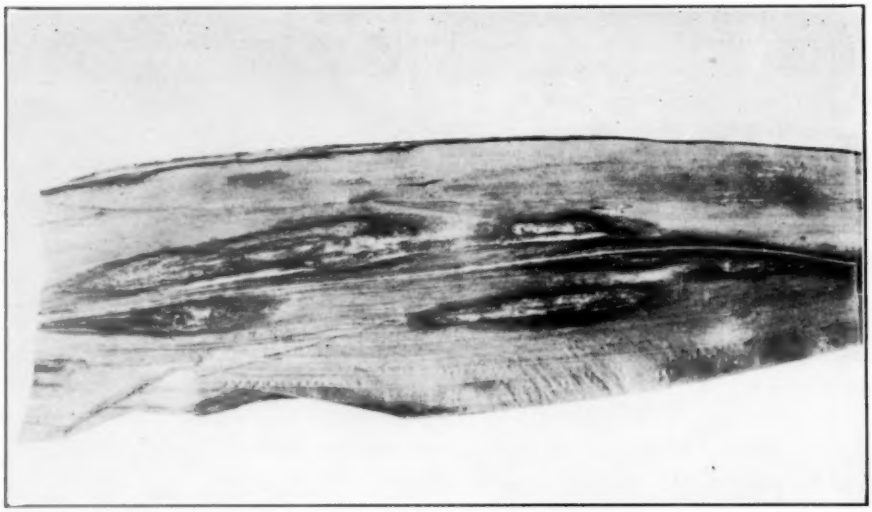

FIG. 1. Lesions caused by *Titaeospora andropogonis* on leaf of sorghum. Natural size.

In many respects the disease (Fig. 1) resembles the leaf blight caused by *Helminthosporium turcicum* Pass. The lesions are elongate elliptic, up to several centimeters long and 1–2 centimeters wide, rather regular in outline, light brown to grayish in the central portion, and bordered by a narrow to broad deep red margin. On the lesions, numerous, small, rough, spherical or subspherical black bodies are usually present, though these are easily brushed off. The occurrence of the black bodies readily distinguishes the disease from leaf blight, and it is due to their presence that the name sooty stripe is suggested. In Manchuria the black bodies appear in late autumn,[1] but in the United States they have been observed as early as July.

[1] Miura, M. Diseases of principal crops in Manchuria. Koshurei Agr. Exp. Sta. of the S. Manchurian Railway Co., Report No. 11. Jan., 1921.

[2] Bain, D. C., and C. W. Edgerton. Two leaf-spot diseases on sorghum and related grasses. Phytopath. 32: 1. 1942.

Thus far, the disease has been reported only in China, Manchuria, and the United States. In Louisiana and Mississippi, it has been found on several varieties of sorghum and on Johnson grass (*Holcus halepensis*).

Miura described the fungus in 1921 and placed it in a new genus, *Ramulispora*. In 1932, however, Tai[3] redescribed it and placed it in the genus *Titaeospora* described by Bubak[4] in 1916. Miura's description of the fungus is as follows: "Conidiophores hyaline, non-septate, slender, 20–35 × 2.5 μ, springing from subepidermal black stroma. Conidiospores cylindrical, flexuose, slender, branched, 4–12 septate, not restricted at septum, 36–100 × 2–4 μ, granular, hyaline. Chamydospores intercalary or terminal, usually several in a chain, yellowish brown, globose or ovate, about 15 μ in diameter, with a few small spinules." The black bodies were referred to as "black bodies of unknown nature." Studies in Louisiana indicate that these black bodies are sclerotia and are possibly formed after sporulation. While Miura placed the fungus in the Melanconiales, recent studies indicate that the fruiting structure is not a typical acervulus but rather a sporodochium which arises immediately below the stomate. Whether Miura's "subepidermal black stroma" is formed before or after sporulation is a matter yet to be determined.

In culture the fungus grows slowly and the mycelium forms a rather dark greenish-gray clump. Conidia are produced in abundance in pinkish thread-like masses in 3–4 days after transferring. Conidia germinate readily within 20 hours. No sclerotia have been observed in culture. Chlamydospores develop readily in bean-pod agar.

Johnson grass and several varieties of sorghum in the greenhouse and field were inoculated in the spring and summer of 1942. Infection was obtained, usually 8–10 days following inoculation, but symptoms as described previously did not appear by September. The fungus was recovered from the small, round to elliptic spots which resulted from the inoculations. The spots attained their maximum size on the sorghum varieties, White Kaoliang, C.P. Special, Standard Broomcorn, and Dwarf Yellow Milo.—DOUGLAS C. BAIN, Department of Botany, Louisiana State University, Baton Rouge, La.

Rhizoctonia Foliage Disease of Hevea brasiliensis.—During a study of Rhizoctonias affecting sugar-beet foliage opportunity was afforded to compare these organisms with a fungus affecting leaves of *Hevea brasiliensis* (H.B.K.) Muell. Arg. Specimens of the affected leaves had been referred to this Bureau by Rolland C. Lorenz, of the Agricultural Experiment Station at Tingo Maria, Peru. The Hevea leaves examined had spots varying from 1 to 10 mm. or more in diameter to blighted areas involving one-half to two-thirds of the leaf blade. The affected areas were commonly surrounded by a narrow, brown border. Within the border a portion of the affected area was white, attributable to the destruction of the parenchyma, the trans-

[3] Tai, F. L. Notes on Chinese fungi. I. Nanking Journ. 2: 171–179. 1932.
[4] Bubak, F. Pilze von verschiedene Standorten. Ann. Mycol. 14: 341–352. 1916.

parent cuticle being left. The white portions of the spots apparently resulted from primary invasions. Successive advances of the fungus from the earlier invaded tissues occurred and were evidenced by collapsed tissue, usually light brown, surrounding the older spots. Brown-tinged, Rhizoctonia-like mycelium was found on the surface of the leaves.

Microscopic examination of the fungus found on the leaves collected at Tingo Maria, Peru, showed it to be of Rhizoctonia type. Distinct rhizomorphs, as reported for *Pellicularia koleroga* (Cke.) Rogers on various hosts, were not present. The hyphae measured 6.5 μ in diameter. The perfect stage of the fungus occurred as grayish white, powdery areas on both sides of the affected leaves. Judging by their abundance, the basidiospores could be a major factor in dissemination.

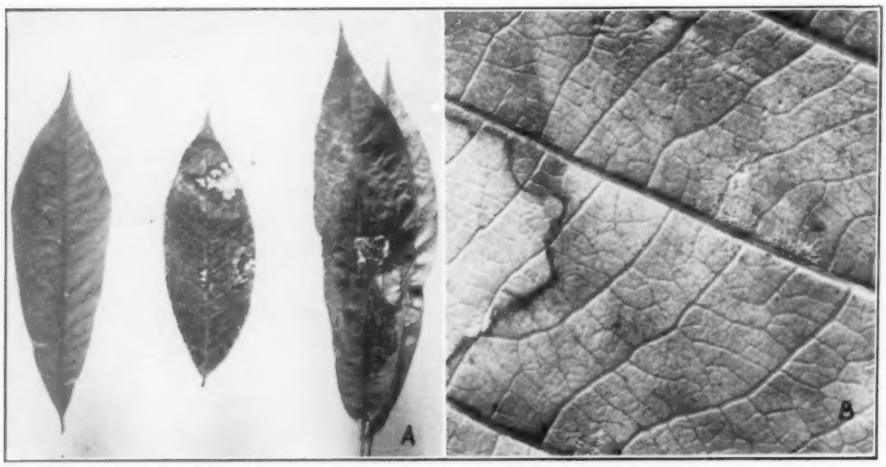

FIG. 1. Rhizoctonia foliage disease of *Hevea brasiliensis*. A. Comparison of a healthy Hevea leaf (left) with infected leaves. The leaf in the center is a naturally infected leaf collected at Tingo Maria, Peru. The leaves at the right were artificially infected with a pure culture of the fungus (R 435) isolated from the material from Peru (0.25×). B. Perfect stage of the organism as produced from an artificial inoculation with the pure culture. In naturally occurring infections, larger areas on both dorsal and ventral surfaces of the leaf may be covered by the spore-producing stage of the fungus (2.5×).

The causal fungus was isolated from a diseased leaf and its pathogenicity tested on greenhouse-grown, rubber-plant seedlings. From a resulting leaf spot the fungus was reisolated and used to inoculate young and old leaves of rubber plants as before. At 21° to 25° C. and with relative humidity at 90 to 100 per cent, infection of young leaves occurred in 5 days (Fig. 1, A) and the perfect stage developed 4 or 5 days later (Fig. 1, B). Leaf penetration was effected from minute infection cushions. No rhizomorphs were found.

On sugar beets under similar environmental conditions foliage infection and formation of the perfect stage of the fungus occurred in 10 to 15 days. The fungus did not cause damping off of sugar-beet seedlings or the rotting of mature roots.

The perfect stage arises from the vegetative mycelium as closely intertwined, more or less dichotomously branched, hyphae tightly adherent to the leaf surface. The basidia are terminal and bear four sterigmata and spores. The sterigmata are approximately the same length as the longest diameter of the basidiospores. Fifty spores obtained from one of the infected leaves of a rubber plant were measured and averaged 8.2×3.7 μ. This spore size and other characters of the fungus conform with *Pellicularia filamentosa* (Pat.) Rogers,[1] as recently proposed. It is to be noted, however, that the perfect stage of the *Rhizoctonia* affecting Hevea foliage is composed of tightly intertwined hymenial cells and is strongly adherent to the surface of the leaf on which it is formed. It is not readily removable. This is in contrast to the loose, rather filamentous structure of the hymenial stage of many other representatives of this admittedly composite species.

The mycelial mat of the fungus when grown on potato-dextrose-agar medium, is brownish black[2] (5 YR 0.7/0.8[3]) and the substrate is heavily darkened as result of the growth of the fungus. The sclerotia are loose textured and, rather uniformly, about 2 mm. in diameter. The perfect stage of the fungus was not observed to occur on this medium.—JOHN E. KOTILA, Pathologist, Division of Sugar Plant Investigations, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture, Beltsville, Md.

Cotton-Leaf-Spot Rhizoctonia and Its Perfect Stage on Sugar Beets.—In connection with determining the pathogenicity to sugar beets of several isolates of *Rhizoctonia*, an organism, isolated by D. C. Neal from a leaf of cotton affected with Rhizoctonia leaf spot, was included.[1]

Under conditions favorable for damping off of seedlings and rotting of older roots of sugar beets the cotton-leaf-spot *Rhizoctonia* was pathogenic on seedlings causing a damping off that started 3 or 4 days after emergence of the seedlings and in 10 days reduced the stand to 61.5 per cent of the control. The fungus was not pathogenic on older roots.

The cotton-leaf-spot *Rhizoctonia* was mildly pathogenic on the foliage of sugar-beet plants kept at 21° to 25° C. and a humidity of 90 to 100 per cent. Infection occurred in 4 days after inoculation but the infected areas remained small. Ten days after inoculation the largest area measured approximately 2×4 cm.

The mycelium of the fungus grew over the surface of the leaf from the sand-corn-meal inoculum as well as from the infected spots and produced the perfect stage of the fungus (Fig. 1), which according to Neal[1] has not been observed on cotton. The hymenial stage appears as a grayish white

[1] Rogers, Donald P. The genus Pellicularia (Thelephoraceae). Farlowia **1**: 96–118. 1943.

[2] Judd, Deane B., and Kenneth L. Kelly. Method of designating colors. Natl. Bur. Standards Res. Paper Rp1239. 1939.

[3] Munsell Book of Color. Revised Text, 1942. Munsell Color Company, Inc., Baltimore, Md.

[1] Neal, D. C. Rhizoctonia leaf spot of cotton. Phytopath. **34**: 599–602. 1944.

powdery growth, readily removable for microscopic examination when wetted with a fine spray of water.

The hymenial cells, basidia, sterigmata, and basidiospores are similiar

FIG. 1. Sugar-beet leaf affected by the cotton-leaf-spot *Rhizoctonia*. The dark, sunken spots are the invaded portions of the leaf. The raised, cottony masses on the leaf surface are vegetative mycelial growths from the inoculum. The perfect stage of the fungus was produced on the leaf surface and appears as a white, powdery growth. The white areas (a) at the top and center of the photograph show the more pronounced development of hymenial structures, but along the veins and rather generally on the leaf surface, other hymenial aggregates occur.

to Rhizoctonias formerly grouped as *Corticium vagum* B. & C. In 50 measurements, spore size averaged $8.8 \times 6.9\ \mu$. The sterigma length was approximately equivalent to the longest spore diameter.

Spore size and other characters place the fungus in the species *Pellicullaria filamentosa* (Pat.) Rogers[2] as recently proposed.—JOHN E. KOTILA, Pathologist, Division of Sugar Plant Investigations, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture, Beltsville, Md.

The Longevity of the Pathogen Causing the Wilt of the Common Bean.—In 1930 the writer[1] pointed out that the pathogen *Corynebacterium flaccumfaciens* (Hedges) Dowson, the cause of the wilt of the common bean, is a bacterium very tolerant to the environment under which it is found and that it can remain viable in dried leaves in the herbarium for at least 5 years. Later Christow[2] in Germany presented evidence which indicated that the pathogen could remain alive for 7 years in bean seed. The 7-year-old seed he used germinated and gave rise in a few cases to wilted plants typical of the disease.

Recent tests by the writer show that *Corynebacterium flaccumfaciens* will live for 24 years in association with bean seed under room conditions. In 1919 a handful of infected White Marrow bean seed was collected and stored in a screw-top glass container, labeled, and placed on a shelf in a laboratory at Cornell University. All these seed had definite yellow varnish-like lesions more or less typical of the wilt disease. After 15 years of storage, experiments were conducted to determine whether or not the pathogen in the seed was still viable and virulent. Dilution plates were made from 3 different seed and in each instance yellow bacteria were isolated that appeared in all respects to be *C. flaccumfaciens*. Inoculations on Red Kidney bean plants growing in the greenhouse proved that the bacteria were pathogenic. At the end of 20 years, in 1939, similar experiments were conducted with like results. In 1943, at the end of 24 years storage, three seed again were tested, but only one yielded viable and virulent cultures, and in this instance the colonies were not so numerous on the plates as they had been in the previous tests. The following year the remaining seed, 10 in all, were tested for living bacteria, but in no case were any organisms found that were able to grow. Twenty-four years appears to be the limit of the pathogen's viability under the conditions in which they were stored. The room was warm and dry, but not excessively so. The bacteria also were further protected underneath the seed coat where they had dried down in the plant juices and were not exposed to the changes, however slight, that went on in the tight glass container. In some respects the pathogen was treated and stored under conditions somewhat similar to those of the Swift method[3] for desiccating and storing bacterial cultures.

[2] Rogers, Donald P. The genus Pellicularia (Thelephoraceae). Farlowia 1: 96–118. 1943.

[1] Burkholder, Walter H. The bacterial diseases of the bean: a comparative study. New York (Cornell) Agr. Exp. Sta. Mem. 127: 36. 1930.

[2] Christow, Alexander. Einige Versuche über die Bakterienkrankheiten bei Bohnen. Phytopath. Ztschr. 7: 537–544. 1934.

[3] Swift, H. F. A simple method for preserving bacterial cultures by freezing and drying. Jour. Bact. 33: 411–421. 1937.

Xanthomonas phaseoli (Smith) Dowson, on the other hand, is not supposed to remain viable in or on the bean seed for much over a year. Rapp[4] states that this pathogen is dead in 2- and 3-year-old seed, and Christow[2] found them non-viable in 7-year-old seed. As far as the writer is aware, nothing has been published on the longevity of *Pseudomonas phaseolicola* (Burkholder) Dowson, the cause of halo-blight of the bean.—WALTER H. BURKHOLDER, Department of Plant Pathology, Cornell University, Ithaca, N. Y.

Phoma terrestris on Sugar-Cane Roots in Louisiana.—Phoma terrestris has been isolated many times during the years 1942 to 1944 from the roots of a number of cane varieties secured from various localities in Louisiana. The same fungus has been isolated also from onion, garlic, sweet clover (*Melilotus indica*), and corn roots as well as from cane, rice, and cotton soils. All isolates were identical in their cultural and morphological characters. The identity of the fungus was verified by Dr. E. C. Tims. The pycnidial stage was readily obtained with all isolates on potato-dextrose agar at room temperatures in subdued daylight.

Inoculations of the cane-root isolates into corn roots were made as follows: yellow corn seeds were germinated under sterile conditions in moist chambers. When the corn roots were from a half to one inch in length, a small piece of agar bearing the actively growing fungus was placed at the tip of each. After inoculation, the plates (moist chambers) were incubated at room temperature and in the dark. After 7 to 15 days, the results were obtained. Cross and longitudinal sections (free-hand) of the affected portions of the inoculated roots were made. It was found that after one week the fungus had usually penetrated the cortex and in some instances the stele as well, although the cortex was the region more generally attacked. All of the isolates of *Phoma terrestris* gave similar results. The tissues affected by the organism turned red-brown. Very often the invaded host cells were entirely filled by the mycelium. The endodermis showed great resistance to the entrance of the fungus mycelium. The roots usually do not get flabby; some get stiff. Other isolates from onion, garlic, and corn, inoculated into corn roots, gave similar results.

These findings demonstrate that *Phoma terrestris* is widely distributed in the soils of the State of Louisiana and most of the isolates penetrated and invaded the tips of the corn roots under laboratory conditions as stated above.—FERNANDO CARVAJAL, 515 Nowlin Ave., Lawrenceburg, Ind.

[4] Rapp, C. W. Aged bean seed, a control for bacterial blight of beans. Science 50: 568. 1919.